THE
LARYNGOSCOPE.

VOL. LXVIII JUNE, 1958 No. 6

STAPES OPERABILITY.

Pathological Indications for Mobilization and Fenestration Surgery.*†

J. BROWN FARRIOR, M.D.,

Tampa, Fla.

In recent years we, as otologists, have learned many techniques for mobilization of the stapes in otosclerotic deafness. Through one technique or another, it is now possible to mobilize a very high percentage of stapes. Even after complete mobilization many of these will show a prompt recurrence of the deafness, because the pathological involvement was beyond the limits of stapes surgery. As otologists we must learn not only the new techniques, but also their pathological limitations.

Initially the technique of the stapes mobilization operation was indirect; that is, all pressures were applied to or through the head and neck of the stapes. With this basic indirect technique, primary hearing improvement was obtained in about one-third of the cases; and when mobilization was technically complete, a high percentage of these maintained the hearing improvement. With this indirect approach, we were mobilizing only those cases with limited pathological involvement, and these pathologically favorable cases tended to maintain their hearing improvement.

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*Presented at the meeting of the Southern Section of American Laryngological, Rhinological and Otological Society, Inc., Memphis, Tenn., Jan. 25, 1958.

†From Southern Foundation of Better Hearing, Tampa, Fla.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Feb. 24, 1958.

Today the indirect approach is largely replaced by a direct surgical attack upon the footplate and the disease process itself. Now the percentage of primary results has almost trebled, and is no longer limited by the strength of the crura; it is limited only by the boldness of the surgeon and a few completely inoperable stapes. With chisel, picks and drills, the enthusiastic surgeon can temporarily mobilize most all stapes. The primary results have increased, but the incidence of re-fixation has also multiplied with this direct attack on the extensively diseased stapes.

If we are to continue to attack these extensively diseased stapes and reduce the incidence of re-fixation, we must proceed with excavation of the footplate and the application of prosthetic crura (Shea[1]), or we must declare more stapes inoperable. Extensive oval window surgery may prove to be worth while, and is worthy of investigation. It may prove to be applicable in certain types of pathological involvement, but there will remain specific pathological limitations.

As we approach the pathological limitations of stapes surgery (indirect, direct, or oval window) we arrive at the pathological indications for the fenestration operation. The fenestration operation has received a wide variety of reputations. By the early 1950s it had reached a standard of perfection that produced a very high percentage of grateful and happy patients, a percentage greater than the ear surgery of 1957; however, some still judge the fenestration operation by its reputation of the early 1940s and regard it only as surgery of the last resort. The fenestration operation is a highly reliable and accurately predictable operation; it remains the master surgical procedure which will restore worthwhile and lasting hearing improvement in a high percentage of suitable cases which are beyond the pathological limits of stapes and oval window surgery.

The thesis of this study is that there are specific pathological indications for stapes surgery and specific pathological indications for fenestration surgery; also, that in stapes surgery there are specific pathological indications for the variations of the indirect and direct surgical approach. To

date otologists have used the chance approach to stapes surgery with the attitude of try, and try again, and some have tended to exclude the fenestration operation until the stapes mobilization has failed, and failed again. Although we can never draw fine lines of differentiation on the operability of the stapes, it is my hope that an appreciation of the tremendous variations in the pathology of the stapes will lead to an understanding of the broader classifications of the favorable, unfavorable and inoperable stapes.

A stapes is called favorable when the anatomy and pathology lend themselves to stapes surgery, and when the prognosis for maintained mobilization and hearing improvement is good.

A stapes is unfavorable when it can be mobilized but the pathology indicates that the prognosis is guarded. A stapes is inoperable when pathologically it cannot be mobilized or, if mobilized, refixation is almost certain. The classification is a surgical opinion of the existing anatomy and pathology, and is not dependent upon technical success or failure.

At the meeting of the American Academy of Ophthalmology and Otolaryngology in October, 1957, I presented preliminary opinions and classifications of the anatomy and pathology of the stapes and their effect upon the operability of the stapes. We are on the threshold of defining the operability of the stapes, based upon the anatomical variations and the type of pathological involvement. This work is here republished with corrections, illustrations, additions and elaborations (Farrior 2a and b).

STAPES OPERABILITY.

The operability of the stapes is dependent upon the type of anatomical development and the pathological involvement. Anatomically the footplate of the stapes may be superficial or in a very deep niche. The first is most favorable for mobilization surgery, while with the deeply placed footplate the crura tend to become involved in the otosclerotic process, making the stapes either unfavorable or inoperable for mobilization surgery.

Pathologically the otosclerosis may involve only the anterior edge of the footplate, or it may involve varying degrees of the footplate, crura, or even the head and neck of the stapes. The simple footplate involvement is very favorable for mobilization surgery, while the complete otosclerotic involvement is inoperable, and is a positive indication for the fenestration operation.

CHART I.

Anatomically variations in the depth of the footplate and angulation of the crura alter the operability of the stapes (see Figs. 1, 2, 3). A superficial footplate with upright crura is the most favorable anatomical development for mobilization surgery.

A.—Depth of the Footplate of Stapes.	Operability (Prognosis)
1. Superficial	Favorable
2. Moderately superficial	Less Favorable
3. Moderately deep	Unfavorable
4. Deep	Unfavorable
B.—Angulation of Crura.	**Operability (Prognosis)**
1. Upright	Most Favorable
2. Downward tilt (free)	Less Favorable
3. Downward tilt (fixed)	Unfavorable
4. Upward tilt	Less Favorable
C.—Strength of Crura.	**Operability (Prognosis)**
1. Weak Crura	Vulnerable
2. Medium Crura	Mod. Vulnerable
3. Strong Crura	Invulnerable
4. Atrophic Crura	Inoperable Footplate

The purpose of this presentation is to demonstrate the varying anatomy and pathology of the stapes and, in so doing, to define the operability of the stapes.

ANATOMICAL VARIABLES.

The position of the footplate may alter the operability of the stapes (see Chart I).

If the footplate of the stapes is very superficially placed, the otosclerotic involvement will usually be thin, and there is no anatomical tendency for the otosclerosis to involve the crura (see Fig. 1). If, on the other hand, the footplate of the stapes is in a deep niche, the crura tend to become tilted downward and thus easily contact the inferior lip of the

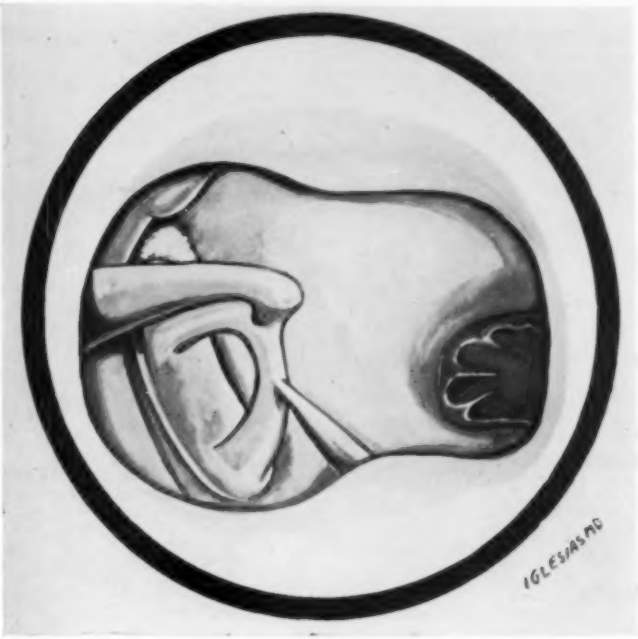

Fig. 1. Anatomically, when the footplate of the stapes is superficially placed and the crura are upright, the otosclerosis tends to remain localized to the level of the footplate. This figure illustrates a superficially placed footplate with upright crura and a small focus of circumscribed anterior footplate otosclerosis (see Chart I). For stapes surgery this is the most favorable anatomical development and pathological involvement.

oval window. This predisposes toward otosclerotic involvement of one or both crura (see Fig. 2), or complete otosclerotic filling of the oval window niche. With a deep footplate, the crura will occasionally tilt upward to contact the Fallopian canal (see Fig. 3). As the pathology progresses the operability becomes unfavorable to inoperable.

The strength of the crura does not greatly alter the operability of the stapes with the direct surgical attack upon the footplate. True, if the crura are weak or friable this predisposes toward their fracture, but too much pressure on the crura is a surgical error rather than the cause of inoperability.

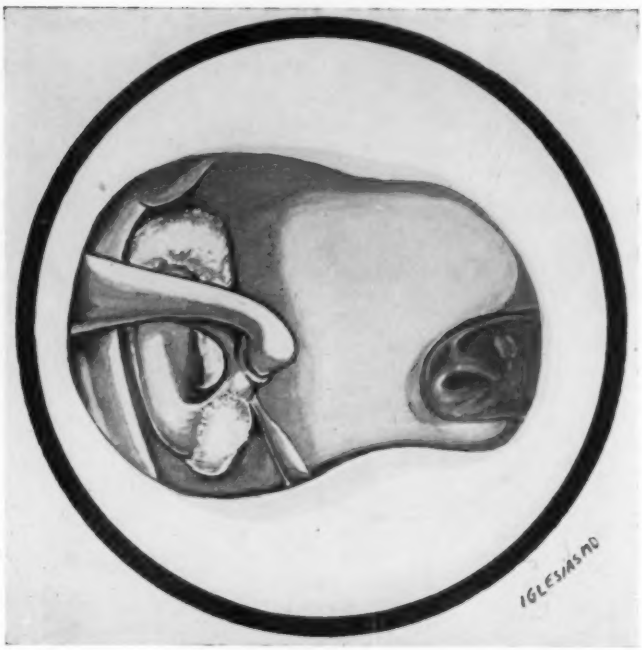

Fig. 2. Anatomically, when the footplate of the stapes is deeply placed in the oval window niche the crura tend to tilt downward and contact the inferior lip of the oval window niche. This contact predisposes toward otosclerotic fixation of the crura, and such crural otosclerosis alters the operability of the stapes. (See Chart I). As re-fixation is prompt, bi-crural otosclerosis is pathologically beyond the operable limits of manipulative operations on the total stapes.

Atrophy of the crura is generally associated with complete footplate otosclerosis which is in itself inoperable. Translucent red crura are the most friable while white crura will withstand the greatest amount of manipulation. As others may wish to include a classification of crural strength, this is included in Chart I.

PATHOLOGICAL VARIABLES.

I. LOCATION OF OTOSCLEROSIS.

The location and character of the otosclerosis greatly alters

CHART II.—PATHOLOGICAL VARIABLES.

The location and extent of the otosclerosis are the most important single factors in determining the operability of the stapes. Originally, I believed that the per cent of involvement of the footplate was the most important factor. At the present time I believe that the thickness of the otosclerosis and involvement of the crura have the greatest effect on operability.

A. Location of Otosclerosis.	Operability.
I. Type of Otosclerosis.	
1. Circumscribed	Favorable (see Fig. 6c)
2. Diffuse	Unfavorable (see Fig. 6d)
II. Footplate Otosclerosis (Fig. 4a, b, c, d).	
1. None	Favorable
2. Anterior Footplate	Favorable
3. Posterior Footplate	Favorable
4. Superior Marginal	Unfavorable
5. Inferior Marginal	Unfavorable
6. 1/4 Footplate Involved	Favorable
7. 2/4 Footplate Involved	Less Favorable
8. 3/4 Footplate Involved	Unfavorable (Thick)
9. Complete Footplate (thin)	Favorable (thin) (see Fig. 6a)
10. Complete Footplate (thick)	Inoperable (thick) (see Fig. 6b)
III. Crural Otosclerosis (see Fig. 5a, b, c, d).	
1. None	Favorable
2. Anterior Crus	Favorable
3. Posterior Crus	Unfavorable
4. Bicrural Anterior and Posterior Crura	Inoperable
5. Apical	Inoperable
6. Cervical	Inoperable
IV. Round Window Otosclerosis.	
1. No Otosclerosis	Favorable
2. Marginal Without Closure	Favorable
3. Partial Closure	Unfavorable
4. Complete Closure	Inoperable for Either Mobilization or Fenestration

the operability of the stapes (see Chart II). Otosclerosis, which alters operability, may involve the footplate of the stapes, the crura of the stapes, the neck of the stapes or the round window.

The two basic types of otosclerosis are circumscribed and diffuse—Fowler.[3] Circumscribed otosclerosis is in general the type which responds best to mobilization surgery, providing the location and anatomy are favorable. Stapes surgery is rarely of lasting value in diffuse otosclerosis which extends over the entire promontory. Eventually we may learn that circumscribed and diffuse otosclerosis are separate diseases.

Fig. 3. Anatomically, when the footplate of the stapes is deeply placed in the oval window niche the crura will sometimes tilt upward to contact the fallopian canal. This contact predisposes toward otosclerosis or post-mobilization scar fixation between the crura and the fallopian canal. (See Chart I).

From clinical experience, it seems that circumscribed anterior footplate otosclerosis is most favorable for mobilization surgery (see Fig. 4a). Posterior footplate otosclerosis is equally favorable, but occurs less frequently (see Fig. 4b). Superior marginal (see Fig. 4c), and inferior marginal (see Fig. 4d), are less favorable, for they tend to involve the sides of the crura. The degree of involvement of the footplate is significant, and its influence upon operability is intimately associated with the thickness of the otosclerosis and crural involvements. Complete otosclerosis of the footplate may still be operable as long as the annulus of the footplate remains well defined and there is no crural involvement; this

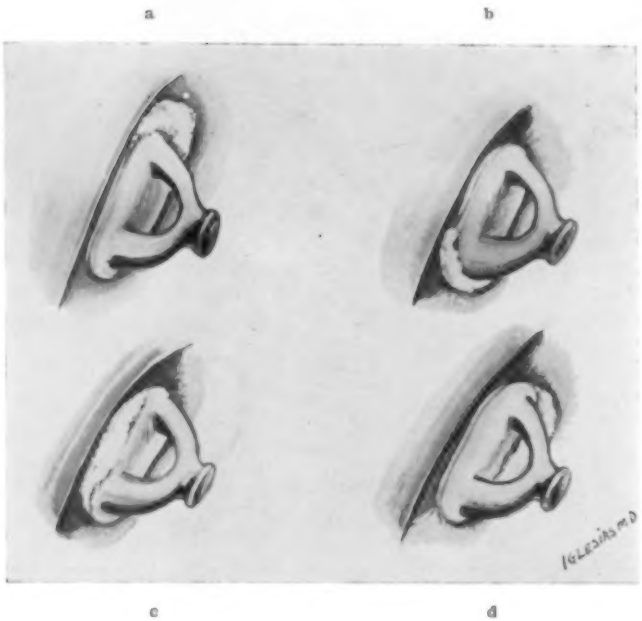

Fig. 4. Footplate otosclerosis. Circumscribed footplate otosclerosis is most favorable for stapes mobilization surgery. The otosclerosis tends to remain at the level of the footplate when the footplate is superficial and the crura do not contact the margins of the oval window niche. Anterior footplate (see Fig. 4b), and superior marginal (see Fig 4c), otosclerosis are present occasionally. Inferior marginal (see Fig. 4d) otosclerosis is frequent, and is usually a continuation posteriorly of anterior footplate otosclerosis (see Chart II).

is found in thin, white otosclerosis (see Fig. 6a). When the otosclerosis is thick enough to obliterate the annulus of the footplate the stapes becomes inoperable (see Fig. 6b).

Otosclerosis may involve one or both crura; as previously stated, crural otosclerosis is most likely to occur in the deeply placed footplate with tilted crura. Anterior crural otosclerosis (see Fig. 5a) is favorable for mobilization surgery, but re-fixation is likely to occur unless an anterior-crurotomy is performed—Fowler.[4] Posterior crural otosclerosis (see Fig. 5b) has not been found as an isolated patholog-

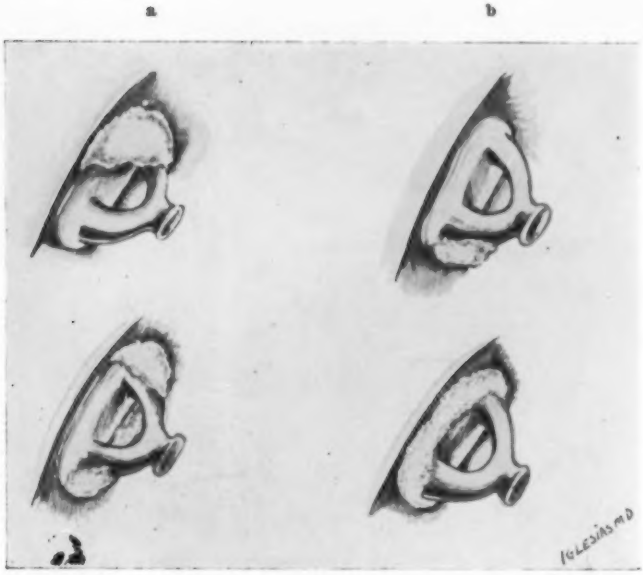

Fig. 5. Crural otosclerosis. Crural involvement by the otosclerotic process greatly alters the operability of the stapes. Anterior crural (see Fig. 5a) otosclerosis is frequent and is an indication for anterior crurotomy and posterior footplate mobilization. Posterior crural (see Fig. 5b) otosclerosis has not been found as an isolated entity. Bi-crural otosclerosis is found with some frequency, and is associated with inferior marginal (see Fig. 5c) or superior marginal (see Fig. 5d) otosclerosis in a deeply placed footplate.

ical entity, but if it does, I should consider a posterior-crurotomy.

Bi-crural otosclerosis has occurred in numerous instances, most frequently with inferior marginal otosclerosis (see Fig. 5c), and occasionally in superior marginal otosclerosis (see Fig. 5d). In each instance there has been re-fixation of the stapes after successful complete mobilization. It is my present opinion that bi-crural otosclerosis is beyond the limits of present day mobilization surgery, and in the suitable candidate is a clear-cut indication for the fenestration operation. In some instances the otosclerotic fixation has extended up the crura to involve the apex of the two crura and the neck

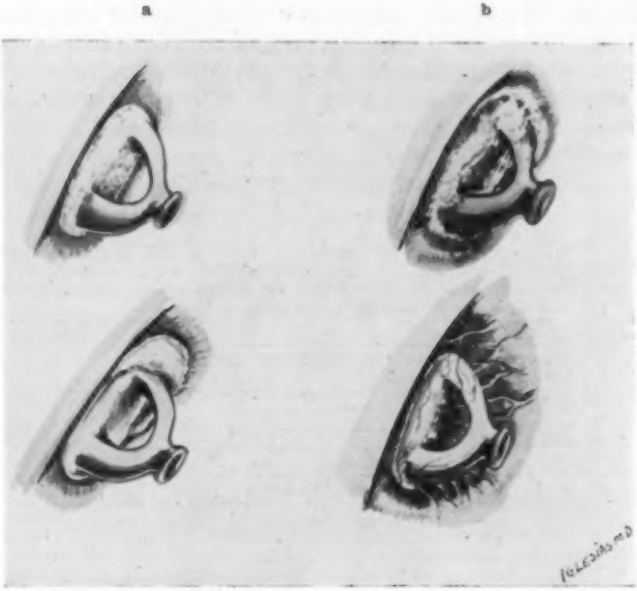

Fig. 6. Character of otosclerosis. The thickness and the color (vascularity) of the otosclerosis alter the operability of the stapes. Complete footplate otosclerosis may still be operable when thin, the annulus is well defined and the crura are not involved (see Fig. 6a); however, when the otosclerosis of the complete footplate and oval window niche becomes thick enough to obliterate the annulus (see Fig. 6b) of the footplate the stapes is then inoperable.

Circumscribed white, avascular, otosclerosis (see Fig. 6c) is most favorable for stapes surgery, while diffuse, red, vascular otosclerosis (see Fig. 6d) will usually re-fix, even after complete mobilization.

of the stapes. Such apical and cervical otosclerosis are beyond the limits of present day mobilization surgery.

Bi-crural otosclerosis, with an area of normal or nearly normal footplate, may prove to be the type of pathology that will respond to mobilization or excavation of the footplate with or without replacing the crura (re-crural or de-crural).

Complete otosclerotic closure of the round window is a contra-indication to mobilization or fenestration surgery. With a partial or almost complete closure of the round win-

dow the patient can still obtain maximum benefits from surgery; however, the long term prognosis is not favorable. Round window closure has occurred independent of stapes fixation; however, in most instances there is associated unfavorable or inoperable pathology of the stapes (diffuse otosclerosis, see Fig. 6d).

CHART III.—PATHOLOGICAL VARIABLES.

The otosclerosis may be thin or thick, white or red, depending upon the activity and vascularity. Circumscribed thin white otosclerosis of the anterior footplate is most favorable for stapes surgery, while diffuse thick red vascular otosclerosis is inoperable.

Character of Otosclerosis.	Operability
V. Thickness of Otosclerosis of Stapes and Oval Window (see Fig. 6a and b).	
1. Very Thin	Favorable
2. Thin	Favorable
3. Moderately Thick	Unfavorable
4. Thick	Inoperable
VI. Color of Otosclerosis (see Fig. 6c and d).	
1. White	Favorable
2. Nearly White	Favorable
3. Pink	Unfavorable
4. Red	Inoperable

V. Thickness of Oval Window Otosclerosis of the Stapes.

The thickness of the otosclerosis alters the operability of the stapes (see Charts III-V). Thin circumscribed otosclerosis is most favorable for mobilization surgery, while thick diffuse otosclerosis is the most unfavorable. As previously stated, as long as the margin of the annulus of the footplate is not obliterated by the disease and the crura are free, the operability is reasonably favorable (see Fig. 6a); however, when the otosclerosis thickens to obliterate the annulus or fill the oval window, the stapes becomes inoperable (see Fig. 6b). This is most frequent in diffuse otosclerosis. In such cases, there is no need to jeopardize inner ear function with excessive footplate surgery when the existing pathology is beyond the limits of stapes surgery, present or future.

VI. Color of Otosclerosis.

The operability of the stapes varies with the color of the

otosclerosis (see Charts III-VI). White (avascular) otosclerosis (see Fig. 6c) is most favorable for lasting results in mobilization surgery, while in red (vascular) otosclerosis (see Fig. 6d) mobilization can be accomplished, but there is almost uniform re-fixation. The vascularity and operability may vary from white, to off-white, to pink, to red. Again, if on a pathological basis the prognosis is most unfavorable, it is absurd to persist in an operation where at best there will be a transient improvement, and where excessive surgery might result in cochlear damage.

THE AUDIOGRAM AND THE LOCATION OF THE OTOSCLEROSIS.

Worthy of note is the effect of otosclerotic involvement upon the audiogram.

Anterior footplate and anterior crural otosclerosis tend to present a flat or ascending air conduction audiogram with nearly normal bone conduction. As the fixation of the footplate progresses the Carhart[5] notch develops. It has long been taught that loss of high tones indicates complete footplate fixation. I have seen a number of cases with flat audiograms and complete footplate fixation; but the flat audiogram persists only as long as good strong free crura are transmitting vibrations to the fixed footplate. When the sides of both crura become fixed, and they are no longer capable of transmitting to the footplate, then there is loss of the high tones (2,000, 4,000 and 8,000), and progression of the Carhart notch, with diminution of the air bone gap at 2,000. This has occurred when crural fixation was associated with complete footplate fixation; but, of greater significance is the fact that this has occurred, in combined marginal and bi-crural otosclerosis when the opposite half of the footplate was a perfectly normal translucent blue; that is, incomplete footplate fixation but complete loss of crural transmission. Subject to subsequent statistical study, it is my present clinical opinion that the audiogram tends to remain flat and speech discrimination good as long as strong free crura are transmitting vibrations to the footplate, even though the footplate itself is completely fixed.

This association of the type of otosclerotic involvement and the high tone loss does not apply to an independent nerve deafness—Farrior.[6] For example, when there is an independent nerve loss for the high tones from one cause (acoustic trauma, presbycusis, etc.), and the patient has otosclerosis, causing the loss of low tones, the association of high tone loss with progression is not true. This type of independent high tone loss does not indicate alteration in the operability of the stapes.

CHART IV.—MISCELLANEOUS PATHOLOGY.

Stapes surgery has revealed a never ending list of miscellaneous lesions which will produce or complicate an otosclerotic type of deafness. This classification is presented for the accumulation of statistical data.

VII.—Mucous Membrane Webs.
VIII.—Fibrous Adhesions.
IX.—Abnormalities of Footplate.
X.—Abnormalities of Anterior Crus.
XI.—Abnormalities of Posterior Crus.
XII.—Abnormalities of Stapedius.
XIII.—Abnormalities of Incus.
XIV.—Abnormalities of Malleus.
XV.—Systemic Diseases.

The cases of combined oval and round window closure have been severely deaf, and audiometrically have shown only a little residual air conduction and bone conduction for the lower tone frequencies; however, such an audiogram does not always indicate round window closure.

MISCELLANEOUS PATHOLOGY.

By clinical observation under the operating microscope we are discovering a never ending list of miscellaneous lesions (see Chart IV), which will simulate the otosclerotic type of deafness or will alter the operability of the stapes. In the past our knowledge of the variables of otosclerosis has been limited to a relatively small number of autopsy specimens—Fowler,[5] Ruedi,[7] Guild,[8,9,10] Eggston and Wolfe,[11] Lindsey,[12] and others. Today, with the operating microscope, and photographic slides of the surgical pathology, the opportunity to learn about the pathology and audiology of otosclerosis is greatly increased.

As otologists learn the detail of the pathology of otosclerosis we add to this never ending list of miscellaneous pathological entities and learn the significance of minute variations in middle ear anatomy and pathology. Mucous membrane webs over and around the stapes are a frequent occurrence, and do not greatly alter the operability of the stapes. I used to destroy these mucous membrane webs to obtain greater visibility of the stapes; however, I believe this results in greater post-operative scar formation, and at present prefer to operate through or around these webs. Dense scar tissue does alter the operability of the stapes, the operability varying with the amount and location of the scar tissue; where

CHART V.—PATHOLOGICAL COMBINATIONS.

The operability of the stapes is determined by the combination of the anatomical and pathological variables. The following are, in general, the order of frequency of these combinations.

1. Anterior footplate.
2. Anterior footplate, anterior crural.
3. Anterior footplate, anterior crural, inferior marginal.
4. Anterior footplate, anterior crural, inferior marginal, posterior crural.
5. Anterior footplate, anterior crural, superior marginal.
6. Anterior footplate, anterior crural, superior marginal, posterior crural.
7. Complete footplate, thin.
8. Complete footplate, thick.

possible, I remove or shunt these fibrous adhesions, but believe that adhesions tend to recur as they do elsewhere in the body.

Schuknecht has reported a case of fixation of the stapedius tendon. Odess reports a case of malleable incus. All of us have seen fixed ossicles in congenital deformities, infections and in arthritis. I have one case with large blue footplate with apparent congenital absence of the annulus of the footplate. This list of miscellaneous pathology will never be complete, and all oddities should be reported. The list of miscellaneous pathology is presented as a basic classification, to which unusual cases can be added.

ACCURATE DIAGNOSIS AND CLASSIFICATION.

Any statistical reporting of results of mobilization surgery

should be based upon the type of anatomical development and the type of pathology present. Only by such a basic classification can we learn which operations, and which techniques, will give lasting results in individual pathological circumstances. This classification of the anatomy, pathology of otosclerosis and miscellaneous pathology is presented as we begin to define the operability of the stapes; degrees of involvement are outlined for coding statistics. The variables are many, and the operability is determined by the combinations of the anatomical and pathological variables (see Chart V). This classification should be changeable; other contributors should make suggestions and corrections, and the opinions on operability of the stapes will naturally vary from year to year as we learn new techniques and new limitations. Certainly the diagnosis "otosclerosis" alone is inadequate for study and future reference. The diagnosis should include a description of the anatomical and pathological findings, and should be complete as:

1. Otosclerosis, circumscribed, thin, white, anterior footplate, 3/4 blue, inferior marginal, favorable.

2. Stapes, fixed, deep, downward tilt.

3. Round window, normal, webs.

4. Hearing, flat audiogram, 45 db. loss, air-bone gap 35, good speech discrimination, good candidate.

ILLUSTRATIVE CASES.

This study is based upon a series of three-dimensional photographs of the surgical pathology of the stapes. More than 850 photographs have been taken in a series of 147 consecutive operations for otosclerosis. The experiences are based upon a series of 326 mobilizations and 724 fenestrations—a total of more than 1,000 operations. The pathology found in selected cases is herein described in detail to illustrate the effect of the variable pathology on the operability of the stapes.

With each illustrative case will be given, first the detailed diagnosis; second, the description of the surgical pathology

in the three dimensional photographs and, finally, the clinical comments on surgical technique and surgical judgment.

The 3-D photographs are taken from the position of the operator, sitting behind the patient looking into the middle ear. The incus is the key anatomical landmark; the pathology will be described in relation to the incus: first, anterior to the incus (around the anterior crus and footplate), and second, posterior to the incus from superior to inferior (left to right). For clarity of presentation all ears are presented as right ears.

OUTLINE OF ILLUSTRATIVE CASES.

Introductory Cases: Case 1—Ideal Pathology; Case 2—Inoperable Otosclerosis; Case 3—Bilateral Otosclerosis; Case 4—Fibrous Adhesions.

Anatomical Positions: Case 5—Downward Tilt; Case 6—Upward Tilt; Case 7—Upward Tilt with Fixation; Case 8—Fallopian Canal Otosclerosis.

Aural Otosclerosis: Case 9—Anterior Crural Otosclerosis; Case 10—Superior Bi-crural Otosclerosis; Case 11—Inferior Bi-crural Otosclerosis.

Character of Otosclerosis: Case 12—Pink Otosclerosis; Case 13—Thin complete footplate otosclerosis; Case 14—Thick complete footplate otosclerosis; Case 15—Inoperable Otosclerosis oval and round window.

Post-Mobilization Pathology: Case 16—Post-mobilization fibrous; Case 17—Probable technical mobilization failure; Case 18—Primary stapes failure from unfavorable pathology; Case 19—Primary stapes failure, inoperable thick footplate.

Fenestration Continuity: Case 20—Footplate mobilization attempted (thick footplate); Case 21—Unfavorable stapes pathology.

INTRODUCTORY CASES.

Case 1. SR—282-F.—Ideal Pathology.

Diagnosis:

1. **Otosclerosis, circumscribed anterior footplate (1/4) thin, white, favorable.**

2. Stapes, fixed, superficial upright.
3. Round window normal.

Description of 3-D Photogroph: (Fig. 1).

A. Anterior to the incus there is an area of thin white footplate otosclerosis; the otosclerosis does not involve the anterior crus.

P. Posterior to the incus, from superior to inferior, are:

1. The normal Fallopian canal.
2. The normal translucent easily seen superficial footplate of the stapes.
3. The upright free strong posterior crus of the stapes.
4. The stapedius tendon.
5. The normal round window.

Comments:

This stapes, with limited pathological involvement of the footplate, was mobilized with the indirect approach by pressure on the incus. The result was lasting.

Case 2. SL—214-MM.—Inoperable Diffuse Otosclerosis.

Diagnosis:

1. Otosclerosis, diffuse, red footplate (3/4), bi-crural, inoperable.
2. Stapes, fixed, deep, downward tilt.
3. Round window closing, nearly complete.

Description of 3-D Photograph: (more than Fig. 6d).

A. Anterior to the incus:

1. Red vascular otosclerosis involves the entire anterior crus to the apex of the crura.
2. The promontory is covered with red vascular otosclerosis.

P. Posterior to the incus, from superior to inferior are:

1. The normal Fallopian canal.
2. A very deep footplate of the stapes, almost invisible.
3. There is a downward tilt of the posterior crus with posterior crural otosclerosis.
4. There is almost complete closure of the round window.

Comments:

Mobilization was easily accomplished by pressure on the incus; however, in thick, red bi-crural otosclerosis of a deep stapes with downward tilt of the crura, the mobilization was of transient value. In almost complete round window closure in active otosclerosis, fenestration would be of transient value, and I have advised against fenestration surgery because of the emotional instability of the patient.

Case 3. SL—283-F.—Bilateral Otosclerosis.

Diagnosis and description:

This is the left ear of Case 1; the diagnosis, anatomy and pathology are almost identical (see Fig. 1).

Comments:

In bilateral otosclerosis the anatomy and pathology of the stapes are similar in type, though they may vary in degree.

In this minimal pathology mobilization was attempted by indirect pressure on the incus; however, when the posterior crus began to redden, indicating an impending fracture, a direct attack was made upon the anterior footplate and complete footplate mobilization obtained.

Case 4. SR—254-Von V.—Adhesions and Otosclerosis.

Diagnosis:

1. Otosclerosis, circumscribed, thin, white anterior footplate with thick fibrous adhesions.
2. Stapes fixed, upright, strong crura.
3. Round window normal.

Description of 3-D Photograph:

This is a favorable stapes, complicated by white fibrous adhesions.

A. Anterior to the incus:

1. Thin, white otosclerosis fixes the anterior margin of the footplate.

P. Posterior to the incus, from superior to inferior, are:

1. The normal Fallopian canal.
2. An area of superficial blue footplate.
3. The anterior portion of the footplate is covered by white fibrous adhesions; the posterior 1/2 of the footplate is normal blue.
4. The posterior crus is strong and upright.
5. The round window is normal.

Comments:

Fibrous adhesions limit the operability of the stapes.

In this case with thin, white anterior footplate otosclerosis and anterior footplate adhesions there was prompt re-fixation of the stapes. Subsequently, an anterior crurotomy and posterior footplate mobilization were performed, giving a lasting result.

ANATOMICAL POSITIONS.

Case 5. SL—255-N.—Downward Tilt.

Diagnosis:

1. Otosclerosis, circumscribed, white anterior footplate, footplate 3/4 blue.
2. Stapes, deep footplate, downward tilt of crura, no posterior crural fixation, unfavorable.
3. Round window normal.

This is an operable but unfavorable stapes with a deep footplate and downward tilt of the crura.

Description of 3-D Photograph: (see Fig. 2).

A. Anterior to the incus:

1. There is circumscribed anterior footplate otosclerosis.

P. Posterior to the incus, from superior to inferior, are:

1. The normal Fallopian canal.
2. The normal blue translucent footplate deeply placed in the oval window niche.
3. The downward tilt of the posterior crus brings it into contact with the inferior lip of the oval window niche. This approximation makes the posterior crus susceptible to otosclerotic fixation by the adjacent otosclerotic involvement. (Similar to Fig. 2 with otosclerosis extending posteriorly adjacent to but not fixing posterior crus).

Comments:

At present this patient has a good result which should last unless there is otosclerotic involvement of the posterior crus.

Case 6. SR—213-A.—Upward Tilt.

Diagnosis:

1. Otosclerosis, circumscribed, thin, white, anterior footplate, footplate not seen.
2. Stapes fixed, very deep, uptilt of crura, contacting Fallopian canal.
3. Normal round window.

This is an operable but anatomically unfavorable stapes.

Description of 3-D Photograph:

P. Posterior to the incus, from left to right are:

1. The normal Fallopian canal.
2. The footplate of the stapes is not visible but in a narrow deep niche.
3. There is marked upward tilt of the crura so that the neck of the stapes is in contact with the Fallopian canal (similar to Fig. 3 with no otosclerosis on Fallopian canal).
4. The round window is normal.

Comments:

This stapes mobilized easily with pressure on the incus. The deep footplate with upward tilt of the crura makes the prognosis guarded in this now successful mobilization (see next case). Although anatomically unfavorable, the result has lasted because there was no crural involvement.

Case 7. SR—269-L.—Upward Tilt Footplate Mobilization and Re-Application of Crura.

Diagnosis:

1. Otosclerosis, diffuse, bi-crural and cervical, with fixation to Fallopian canal and obliteration of 1/2 of footplate.

2. Stapes fixed, very deep, inaccessible footplate, upward tilt.
3. Round window patent but marginal otosclerosis.

This is an extensive unfavorable otosclerosis.

Description of 3-D Photograph:

A. Anterior to the incus there is:
   1. Red otosclerotic involvement of the promontory.

P. Posterior to the incus, from superior to inferior, are:
   1. Otosclerosis of the Fallopian canal.
   2. Otosclerotic fixation between the Fallopian canal and the neck of the stapes. (Cervical otosclerosis—more than Fig. 3).
   3. Red vascular otosclerosis of the promontory.
   4. Normal round window.
   5. Footplate of the stapes is so deep that it is invisible; the resultant upward tilt of the crura predisposed toward the otosclerotic fixation of the neck.

Comments:

The red vascular otosclerotic fixation between the Fallopian canal and the neck of this deeply placed stapes, with upward tilt and fixation of both crura, makes this an inoperable stapes by currently accepted mobilization techniques. As this 54-year-old male presented 85 db. loss and was not suitable for fenestration surgery, the footplate of the stapes was investigated. The crura of the stapes were fractured and retracted downward to expose the footplate. (The anterior footplate, anterior crural, inferior marginal and posterior crural otosclerosis were more extensive than Fig. 5c). Approximately one-half of the footplate was normal blue. This central portion of the footplate was mobilized with the chisel. The remnant of the stapes still attached to the incus was swung back into position over the mobile central portion of the footplate. This operation was done on September 10, 1957. On February 7, 1958 (5 months post-operative), the patient still poossessed a 42 db. hearing improvement to the 43 db. hearing level.

Such bi-crural otosclerosis is beyond the limits of accepted methods of mobilization surgery, and as previously stated, required some type of oval window surgery with or without the application of prosthetic crura.

Shea advocates excavation of the oval window and the aplication of a vein graft and prosthetic crura or re-application of a uni-pod stapes.

As in this case, at the present time, I feel that such oval window surgery should be reserved for those cases which are no longer suitable for the fenestration operation.

Case 8. SR—308—Fallopian Canal Otosclerosis.

Diagnosis:

1. Otosclerosis, circumscribed, red, Fallopian canal to anterior crus, footplate normal.
2. Stapes fixed, upright, deep.
3. Round window normal.

Description of 3-D Photograph:

P. Posterior to the incus from left to right are:

1. The Fallopian canal.
2. On the inferior border of the Fallopian canal there is a red prominence of otosclerotic bone projecting downward to fix the superior surface of mid-portion of the anterior crus (Similar to Fig. 3 but not localized to anterior crus).
3. Deep to the bony prominence there is a normal blue, deeply placed footplate.
4. The posterior crus and stapedius tendon are not involved.
5. The chorda tympani nerve and round window are normal.

Comments:

The superior otosclerotic fixation external to a normal footplate is rare. Because of the anterior crural fixation an anterior crurotomy was attempted; however, in the process the stapes was completely mobilized, making crurotomy impracticable. At present, the hearing is good.

CRURAL OTOSCLEROSIS.

Case 9. SR—272—Anterior Crural Otosclerosis.

Diagnosis:

1. Otosclerosis, circumscribed, thick, pink anterior crural, footplate 3/4 blue, favorable.
2. Stapes fixed, moderate superficial upright, strong crura.
3. Round window normal.

Description of 3-D Photograph: (Less than Fig. 4a).

A. Anterior to the incus:

1. Thick, white, otosclerosis surrounds the anterior footplate and anterior crus.

P. Posterior to the incus, from superior to inferior, are:

1. The normal Fallopian canal.
2. A moderately deep, uninvolved area of the footplate of the stapes.
3. A strong, free, upright posterior crus.
4. A normal round window.

Comments:

In thick, white anterior footplate and anterior crural otosclerosis, the prognosis is favorable, with Fowler's anterior-crurotomy and complete posterior footplate mobilization of the stapes.

Case 10. SR—283-SS.—Superior Bi-Crural Otosclerosis.

Diagnosis:

1. Otosclerosis, circumscribed, red, thick, superior marginal, bi-crural, inferior 1/2 of footplate blue.
2. Stapes, fixed footplate, deep, crura upright.
3. Round window normal.

Description of 3-D Photograph:

P. Posterior to the incus, from superior to inferior, are:

1. The normal Fallopian canal.
2. Red vascular otosclerosis along the entire superior margin of the footplate replacing the annulus, fixing the length of the footplate, and fixing the superior edge of the anterior and posterior crus and filling the superior 1/2 of the oval window niche (Fig. 5d).
3. The inferior half of the footplate is normal translucent blue.
4. The inferior margin of the annulus is normal.
5. The round window is normal.

Comments:

It is easy enough to mobilize such a stapes, but there is usually prompt re-fixation with thick, red superior marginal, bi-crural otosclerosis. Audiometrically, this bi-crural otosclerosis shows a sloping air-conduction curve with loss of high tones.

Because of the marked narrowing of this deep oval window niche, and the fact that this 22-year-old female is suitable for fenestration surgery, I have advised against further oval window surgery, and have encouraged the patient to have a fenestration operation.

Case 11. SL—225-MH.—Inferior Bi-Crural Otosclerosis.

Diagnosis:

1. Otosclerosis, circumscribed, white anterior footplate, inferior marginal, bi-crural, 3/4 of footplate blue, unfavorable.
2. Stapes fixed, deep footplate, downward tilt of crura.
3. Round window normal.

Description of 3-D Photograph:

A. Anterior to the incus there is:

1. A wide area of white anterior footplate and anterior crural otosclerosis.

P. Posterior to the incus, from superior to inferior, are:
1. The normal Fallopian canal.
2. An area of normal blue footplate deeply placed in a narrow niche.
3. The normal superior and inferior margins of the footplate.
4. The downward tilt of the crura brings the crura into contact with the inferior lip of the oval window; there is white otosclerotic fixation of the inferior border of the posterior crus (see Fig. 5c).
5. The round window is normal.

Comments:

This bi-crural fixation of the deeply placed stapes with downward tilt of the crura is, to me, beyond the limits of our present day mobilization surgery. In this case, and others, re-fixation has been prompt. This is the type of case which might respond to mobilization of the footplate combined with an anterior and posterior crurotomy, and either the insertion of some type of non-osteogenic prosthetic crura, or a Type IV tympanoplasty. As the case is clinically suitable, I have advised a fenestration operation.

CHARACTER OF OTOSCLEROSIS—COLOR AND THICKNESS.

The color of the otosclerosis is dependent upon the vascularity and the activity of the otosclerotic process; the circumscribed white, inactive otosclerosis is difficult to mobilize, but does not tend to re-fix, while the red vascular otosclerosis is easy to mobilize but tends toward prompt re-fixation.

White, nearly white, and red otosclerosis has been illustrated by previous cases.

Case 12. SL—263-H.—Diffuse Pink Otosclerosis.

Diagnosis:
1. Otosclerosis, diffuse, pink anterior footplate, unfavorable.
2. Stapes fixed, deep, upright, crura not fixed.
3. Round window, patent, marginal otosclerosis.

Description of 3-D Photograph:

A. Anterior to the incus:
1. There is pink otosclerosis fixing the anterior footplate and extending over the promontory.

P. Posterior to the incus, from superior to inferior, are:
1. Pink otosclerosis involving the Fallopian canal.
2. A deeply placed almost invisible footplate.

3. Upright crura which do not touch the margins of the oval window (similar to Fig. 3 without contact).
4. Pink otosclerosis extending over the promontory (similar but less than 6d).
5. Patent round window with marginal round window otosclerosis.

Comments:

In general re-fixation is prompt in such pink otosclerosis; however, this case has maintained hearing improvement. This lasting improvement probably occurred because the crura of the stapes were upright, and were not involved in the otosclerotic process. In stapes surgery it is the possible combinations of favorable and unfavorable anatomy and pathology which determine operability (see Chart V).

Case 13. SL—247-A.—Complete Thin Footplate Otosclerosis.

Diagnosis:

1. Otosclerosis, circumscribed, thin, white, complete footplate, annulus discernable.
2. Stapes fixed, superficial, crura upright, free.
3. Round window normal.

Description of 3-D Photograph:

P. Posterior to the incus, from left to right, are:

1. The normal Fallopian canal.
2. The footplate is superficial and completely replaced by the otosclerotic process. The margins of the footplate are thin so that the annular margin of the footplate is visible superiorly and inferiorly (see Fig. 6a).
3. The crura are strong upright and do not contact the margins of the oval window.
4. The round window is normal.

Comments:

This appeared to be an inoperable footplate; however, with manipulation at the annulus with the chisel, complete footplate mobilization was obtained, and the patient has a lasting hearing improvement.

Six months post-operatively, this patient states that her hearing has remained good, and the audiogram shows a 30 db. hearing improvement to the 25 db. level.

Case 14. SL-237-S.—Thick Complete Footplate Otosclerosis.

Diagnosis:

1. Otosclerosis, diffuse, red, thick, complete footplate, with filling of the oval window niche.
2. Stapes fixed, deep footplate, crura free.

3. Round window normal.

Description of 3-D Photograph:

P. Posterior to the incus, from superior to inferior, are:

1. A normal Fallopian canal.
2. A deeply placed footplate completely covered with thick, red, vascular otosclerosis which fills the oval window niche to apex of the crura of the stapes (see Fig. 6b).
3. The round window is normal.

Comments:

Thick, red, complete footplate otosclerosis is inoperable and is a pathological indication for the fenestration operation. I do not believe that oval window surgery will ever be applicable when the otosclerosis is so thick that the margins of the footplate are obliterated, and there is no remaining normal footplate.

When exploration revealed the above described pathology, the operation was discontinued and a fenestration advised. It is not necessary to fracture the crura, or to drive a chisel into the footplate, to know that mobilization would be a failure.

Case 15. FL-718-B.—Inoperable Otosclerosis, Oval and Round Window.

Diagnosis:

1. Otosclerosis, diffuse, red, anterior footplate, inferior marginal, bi-crural, promontory, oval window.
2. Stapes fixed, deep footplate, downward tilt.
3. Round window, complete closure.

Description of 3-D Photograph:

P. Posterior to the incus, from superior to inferior, are:

1. The normal Fallopian canal.
2. A very deep, almost occluded, footplate of the stapes.
3. Downward tilt of the crura of the stapes, with complete otosclerotic fixation of one-half of the posterior crus (worse than Fig. 6d with complete round window closure).
4. Red, vascular, otosclerosis of the promontory.
5. Complete slit-like closure of the round window.

Comments:

A primary fenestration was advised because of the positive Schwartze sign, but surgery was discontinued when exploration revealed the complete round window closure. A fenestration may be of value if even a fraction of the round window is patent. A hearing aid is indicated in complete round win-

dow closure. Had the oval window not been completely closed, I would have proceeded with the fenestration operation.

POST-MOBILIZATION PATHOLOGY.

Re-fixation of the stapes, or primary mobilization failures, may result from either technical or pathological causes. Re-fixation of the stapes may result from inadequate surgery, but more frequently re-fixation occurs in unfavorable or inoperable anatomical and pathological circumstances. This is the cause for re-fixation after complete mobilization. Inadequate surgery includes incomplete mobilization, and failure to do an anterior-crurotomy in anterior crural otosclerosis.

Primary mobilization failures may result from inexpert surgery, or improper surgical attack; more frequently, primary failures occur to the reasonably competent surgeon, because the pathology present was beyond the limits of stapes mobilization surgery. A primary mobilization failure of inexpert surgery may convert a thoroughly operable stapes into an unfavorable or inoperable stapes.

Re-operation on the stapes is rarely indicated because of probable primary unfavorable pathology and secondary scar formation. Re-operation on the stapes is indicated only when the primary pathology and anatomy were found to be favorable. Re-operation on the stapes should never be performed as a "solo" operation, for too often the re-operated stapes is found to be inoperable; therefore, in re-operation of the stapes one should be prepared to do a "fenestration in continuity" should the stapes prove to be pathologically inoperable.

Case 16. SR—65-FR.—713 EF.

Diagnosis:

1. Post-mobilization fibrosis, severe.
2. Otosclerosis, circumscribed, anterior footplate, anterior crural.
3. Stapes fixed, deep footplate, downward tilt.
4. Round window normal.

Description of 3-D Photograph:

This is a post-operative mobilization primary failure in which we are performing a fenestration operation and have exposed the entire incus.

P. Posterior to the incus, from superior to inferior, are:

1. The Fallopian canal covered with scar tissue.
2. Firm fibrous adhesions occlude the view of the deep footplate of the stapes and bind the stapes and stapedius tendon to the Fallopian canal and incus to the chorda tympani nerve, tympani membrane and promontory.

Comments:

These fibrous adhesions of a deeply placed stapes, with downward tilt of the crura, make this an inoperable stapes and is a pathological indication for the fenestration operation.

Case 17. S—67-F.—715-A—Probable Technical Failure.

Diagnosis:

1. P. O. mobilization, re-fixation.
2. Otosclerosis, anterior footplate, anterior crural, inferior marginal, firm fibrous adhesions.
3. Stapes fixed, deep, downward tilt.

Description of 3-D Photograph:

P. The incus has been dislocated, from superior to inferior, are:

1. The ampulla of the horizontal canal.
2. The Fallopian canal.
3. The deeply placed footplate.
4. Downward tilt of the posterior and inferior crus of the stapes, otosclerosis fixing 1/2 of the anterior crus (see Fig. 5a).
5. Fibrous adhesions fixing the stapes.

Comments:

This re-fixation of the stapes is probably a technical failure, for the patient has a good mobilization on the opposite ear, and in bilateral otosclerosis the pathology in the two ears is similar. Anterior crurotomy may have prevented re-fixation. In the opposite ear, the patient has maintained a 30 db. hearing improvement to the 15 db. level for 18 months. In the operated ear under discussion the patient obtained a 25 db. gain to the 30 db. level with prompt loss of the gain. As the secondary anatomy and pathology were unfavorable, a fenestration was performed yielding a 35 db. gain to the 20 db. level.

In cases that have had stapes mobilization on both ears, if they have obtained one good result they are anxious to have a fenestration on the opposite ear; however, if both mobilizations have been a failure, they are reluctant to try any further surgery. If the pathology is found to be unfavorable for

stapes surgery in the first ear, I advise a fenestration on this ear before exploring the stapes on the opposite ear. If the pathology was favorable in the first ear and the mobilization was a failure for technical reasons, a mobilization is performed on the opposite ear.

Case 18. SL—276-FL.—721-MD.—Unfavorable Stapes.

Diagnosis:

1. P. O. stapes primary failure.
2. Otosclerosis, circumscribed, anterior footplate, anterior crural, inferior marginal, posterior footplate, footplate 1/5 blue.
3. Stapes, footplate deep, crura downward tilt, fractured, bound with adhesion.
4. Round window normal.

Description of 3-D Photograph:

1. The incus, scar tissue and crura of the stapes have been removed, exposing the oval window niche and footplate; from superior to inferior are:
   1. The ampulla of the horizontal canal.
   2. The Fallopian canal.
   3. The deeply placed footplate of the stapes showing anterior and posterior footplate otosclerosis involving the stumps of the crura. The circumscribed white otosclerosis extends onto the promontory for about 1 mm. (more than Fig. 5c).

Comments:

The stapes mobilization was a primary failure because of the crural and footplate otosclerosis. Because of the small central area of blue footplate (1/5) some might advise excavation of the oval window and the application of prosthetic crura. As a secondary operation, in a suitable candidate, I prefer the more reliable and predictable fenestration.

Some surgeons justify multiple stapes operations on the patient's temporary objections to a fenestration. Unless the secondary surgical procedure is reasonably predictable, I advise a hearing aid.

Case 19. SL—99-FL.—720-EK.—Inoperable Footplate.

Diagnosis:

1. P. O. stapes primary failure.
2. Otosclerosis, diffuse, thick, pink, complete footplate, annulus obliterated, crura atrophic.
3. Stapes fixed, superficial crura upright, weak, no fibrosis.
4. Round window normal.

Descroption of 3-D Photograph:

1. A fenestration is being performed, the incus has been removed, from superior and inferior are:

1. The ampulla of the horizontal canal.
2. The Fallopian canal.
3. The footplate of the stapes is completely replaced by thick, pink, otosclerosis with obliteration of the annulus; there is no scar formation.
4. The crura are atrophic.
5. The round window is normal.

Comments:

At the primary mobilization the crura of this inoperable stapes were fractured. This thick footplate is beyond the operable limits of any known type of oval window surgery. The fenestration was performed with a 26 db. gain to the 22 db. level.

FENESTRATION IN CONTINUITY.

Case 20. FR—710-H.—Inoperable Stapes.

Diagnosis:

1. Otosclerosis, circumscribed, complete footplate, white, flat, 1+ thick, annulus upright, strong.
2. Stapes fixed, superficial footplate, crura upright, strong.
3. Normal round window.

Description of 3-D Photograph:

P. Posterior to the incus from superior to inferior are:

1. The normal Fallopian canal.
2. The footplate is broad, white, superficial, with the annulus almost obliterated (between Fig. 6a and b).
3. The posterior crus is strong and upright.
4. The round window is normal.

Comments:

Because of the severity of the deafness (80 db.), a fenestration was advised, anticipating an inoperable stapes. When the exploratory tympanotomy revealed the anatomically favorable strong upright crura, and the anatomically favorable superficial footplate with white otosclerosis, efforts were made to mobilize the footplate, even though there was complete footplate otosclerosis with beginning obliterations at the annulus. The chisel was used completely around the annulus of the footplate, but the footplate could not be mobilized. A fenestration was performed in continuity, with resultant 37 db. gain.

In other instances, I have prepared for a fenestration and done a mobilization when the stapes proved to be operable.

A fenestration is performed in continuity when the pathology is found to be beyond the limits of mobilization surgery. The decision to perform a fenestration in continuity is based, not upon the blind audiometric failure, but upon the microscopic fact that the footplate is not mobile, and that the pathology is beyond the limits of stapes surgery.

Case 21. FR—717-BS.—Unfavorable Stapes.

Diagnosis:

1. Otosclerosis, white, circumscribed, anterior footplate, anterior crural, inferior marginal, posterior crural, unfavorable.
2. Stapes fixed, deep, downward tilt.
3. Round window normal.

Description of 3-D Photograph.

A. The incus has been dis-articulated; from superior to inferior are:

1. The ampulla of the horizontal canal.
2. The normal Fallopian canal.
3. The deeply placed footplate of the stapes.
4. There is a downward tilt of both crura; white otosclerosis is seen to involve one-third of the anterior crus, extend along the entire inferior margin of the footplate and involve the posterior crus (see Fig. 5c).

Comments:

When exploratory tympanotomy revealed this unfavorable pathology we proceeded with a "fenestration in continuity" without attempting mobilization. The patient and the operating staff had been previously prepared, because of the sloping air-conduction audiogram with the disproportionate loss of high tones. It would have been easy enough to mobilize this stapes, but in my present clinical opinion, the result would have been temporary, because of the bi-crural otosclerosis.

Some surgeons advise that a stapes mobilization should always be tried first. A thesis of this study is that this trial and failure approach should be replaced by sound surgical judgment, based upon the clinical interpretation of the existing pathological circumstances.

CLINICAL COMMENTS.

1. Stapes mobilization is the operation of choice in early and moderate circumscribed otosclerosis.

2. In bilateral otosclerosis the anatomy and pathology of the stapes are similar in type, though they may vary in degree.

3. If the stapes mobilization on the first ear reveals favorable surgical pathology and favorable surgical anatomy, a mobilization is advised on the opposite ear.

4. If the stapes mobilization has been primarily successful in the presence of unfavorable pathology, the patient is advised of these findings and that he will probably need a fenestration. While the hearing is still good and the morale high, he is given a tentative date for the subsequent fenestration (interval about three months).

5. (a) If the first stapes mobilization was a failure, the surgical pathology is unknown, and the operated ear is the poorer ear, a fenestration operation is advised on this ear (see secondary mobilization surgery).

(b) If at the time of the fenestration, examination reveals that the stapes was primarily operable, subsequently a stapes mobilization is performed on the opposite ear.

6. If the first stapes mobilization is a failure, the surgical pathology unknown, and the unoperated ear is the poorer ear, an exploratory tympanotomy is done on the unoperated ear and a mobilization or fenestration done, depending upon the pathological findings.

7. A positive Schwartze sign is probably a contra-indication to mobilization surgery; certainly it is a contra-indication to a "solo" mobilization.

8. In mobilization surgery crural otosclerosis is an indication for crurotomy to prevent re-fixation.

9. Bi-crural otosclerosis is probably a contra-indication to mobilization surgery, although in the severely deaf some improvement may result from anterior and posterior crurotomy and mobilization or excavation of an operable footplate, with or without the application of prosthetic crura.

10. Bi-crural otosclerosis is believed to be indicated clinically by otosclerotic loss of the high tones.

11. Complete otosclerosis of the footplate may still present a flat audiogram if there is no crural fixation, at least until the otosclerosis becomes too thick or the crura too weak to transmit sound.

12. Bi-crural otosclerosis with round window closure is indicated clinically by the very severe associated "nerve" loss.

RE-FIXATION AND SECONDARY SURGERY.

13. Re-fixation of the stapes may result from inadequate surgery.

14. Most instances of re-fixation of the completely mobilized stapes result from unfavorable surgical anatomy and surgical pathology.

15. Re-operation on the stapes is indicated only in the presence of the most favorable anatomy and pathology, and primary incomplete mobilization.

16. Re-operation on the stapes is contra-indicated in unfavorable anatomy and pathology, unfavorability includes both the primary otosclerosis and the secondary post-mobilization cicatrization.

17. At the present time, in clinically suitable cases, I favor the fenestration operation over anterior and posterior crurotomy, and mobilization of the footplate alone or excavation of the footplate and the application of prosthetic crura.

18. Many stapedial footplates are so involved that they cannot be mobilized or excavated, and are an unequivocal indication for the fenestration operation in clinically suitable cases.

19. Adequate exposure and adequate magnification are advocated to:

1. Give maximum opportunity for varied surgical attack on the variable anatomy and pathology of the stapes.
2. To determine the pathological limitations of the stapes surgery.
3. To determine the pathological indication for fenestration surgery.

SUMMARY.

1. There are specific pathological indications for stapes surgery and there are specific pathological indications for fenestration surgery.

2. The operability of the stapes is based upon the type of anatomical development, the degrees of pathological involvement and the possible combinations of these variables.

3. There is often a direct correlation between the severity of the deafness, the pathological involvement and the operability of the stapes.

4. A basic classification of the anatomy and pathology is presented; degrees of involvement are presented for coding of anatomy and pathology and the classification of surgical results.

5. Any classification of results of stapes surgery should be based upon the type of anatomical development, and the type of pathological involvement. In this way we will ultimately define the operability of the stapes.

6. Current opinions are expressed relative to the favorable, unfavorable, and inoperable stapes, based upon the anatomical development and pathological involvement.

7. The variable operability of the stapes is described in a series of illustrative cases.

8. The stapes mobilization operation is a reliable surgical procedure in favorable anatomical and pathological circumstances.

9. Stapes surgery has enough advantages to make it worthy of a trial in less favorable anatomical and pathological circumstances.

10. The fenestration operation is indicated when the anatomical development and pathological involvement are beyond the limits of reasonably predictable stapes surgery.

11. If the stapes surgeon strives to obtain the maximum percentage of primary mobilization results in all types of

pathological involvement, he will jeopardize the functions of many inner ears with little gain in the percentage of cases with lasting hearing improvement. We are learning the limits of successful mobilization surgery.

12. Unfavorable pathology is the most frequent cause for re-fixation after complete stapes mobilization.

13. Incomplete stapes mobilization or inexpert surgery may convert an operable stapes into an inoperable stapes.

14. Re-operation of the stapes is rarely indicated unless the first surgery revealed favorable pathology and the mobilization was incomplete.

15. True, the inoperability of the stapes cannot be determined without microscopic inspection of the pathology, but inoperability may be suspected if there is a positive Schwartze, a high tone loss, or if the patient is very hard of hearing (50 db. or more).

16. The inoperable stapes is a firm pathological indication for the fenestration operation in a clinically suitable case. Inoperability of the stapes may result from extensive otosclerosis, vascular otosclerosis, post-mobilization cicatrization, adhesive processes, or a combination of these, with unfavorable anatomical development.

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To be published. (When I presented this material at the Academy I learned that Ruedi was conducting a parallel study, and that many of his ideas are similar. I have been unable to obtain a copy of his paper and this reference is included here for completeness.)

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MICHIGAN MAN WINS HEARING SOCIETY AWARD.

Stuart L. Edmonds, Midland, Mich., was recently named winner of the American Hearing Society's Kenfield Memorial Scholarship, an award made annually to a prospective teacher of lipreading. Mr. Edmonds is the first man to receive the honor since establishment of the scholarship fund in 1937.

Mr. Edmonds has a bachelor's degree in speech correction, University of Michigan, and a master's degree in education, Eastern Michigan College of Education, Ypsilanti. He has taught speech correction in his home State for five years.

He is currently employed as field service consultant for the Michigan Association for Better Hearing, a job which has acquainted him with the urgent need for teachers of lipreading. Through funds from the scholarship, he plans to take a course at Northwestern University in Methodologies for Teaching Speech Reading.

SUPRAGLOTTIC SUBTOTAL LARYNGECTOMY AND RADICAL NECK DISSECTION FOR CARCINOMA OF THE EPIGLOTTIS.

JOSEPH H. OGURA, M.D.,*†

St. Louis, Mo.

The presently accepted method for the surgical treatment of cancer of the epiglottis is total laryngectomy and radical neck dissection;[1-8] yet, in certain instances of carcinoma of the epiglottis, the surgeon must wonder whether sacrifice of the entire larynx and the commission of the patient to permanent tracheostomy is entirely necessary.

The objective of the surgeon should be not only adequate removal of the lesion but also preservation of laryngeal physiology. Peroral amputation of the epiglottis in the absence of nodal metastases has never received popular acceptance, since obviously a wide margin of safety could not be accomplished. Although partial laryngectomy with preservation of cordal function is possible, it requires the creation of a large pharyngostoma. The attendant dysphagia, drooling of saliva and multiple subsequent operations to effect closure have influenced surgeons into taking the easier course of total laryngectomy.

Supraglottic subtotal laryngectomy and radical neck dissection as a one-stage operation for carcinoma of the epiglottis was developed in an attempt to conserve the function of the larynx. In South America the procedure is known as the partial horizontal laryngectomy.[9,10] The procedure described here differs from the South American method in that primary closure is performed, rather than to create a pharyngostoma which necessitates a second operation at a later date.

The rationale of the proposed surgical procedure is based

*Presented at the American Academy of Ophthalmology and Otolaryngology, Chicago, Ill., Oct. 16, 1957.

†Department of Otolaryngology, Washington University School of Medicine, St. Louis, Mo.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication April 29, 1958.

on embryological, anatomical, clinical and pathological principles. The embryological studies of Most[11] and the work of Rouviere[12] show that the lymphatic pathways from the supraglottic area are independent of those from the true cords and the subglottic areas. The supraglottic portion of the larynx develops from the primitive pharynx while the glottis and subglottic area are derived from the superior extremity of the tracheal tube. The ventricular bands, epiglottis and the

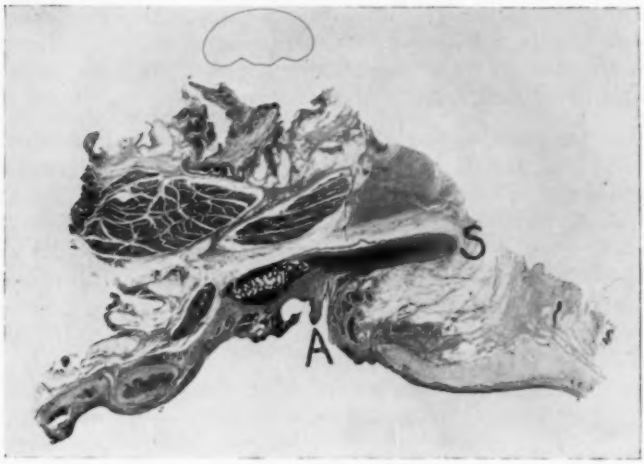

Fig. 1. The relation of the anterior commissure to the thyroid cartilage. Note the inferior position of the anterior commissure—A. From the superior border of the thyroid cartilage; S. Low power vertical section.

anterior part of the aryepiglottic fold arise from the fourth branchial arch. The separate lymphatic collecting pathways from the supraglottic and the cordal with subglottic area would suggest that the two sites could be divided from one another without violating sound principles of cancer surgery.

The preepiglottic space anatomically is confined between the hyoid and anterior commissure of the true vocal cords, shaped like a pyramid with the base up, and bounded laterally by the hyoepiglottic and hyothyroid and thyroepiglottic mem-

branes and ligaments. The apex of this cone-shaped structure ends just superior to the anterior commissure of the true vocal cord, and is less than one-half the distance between the superior notch and the inferior border of the thyroid cartilage (see Fig. 1).

Studies of larynges removed for primary malignant tumors of the epiglottis may show an invasion of the tumor into the

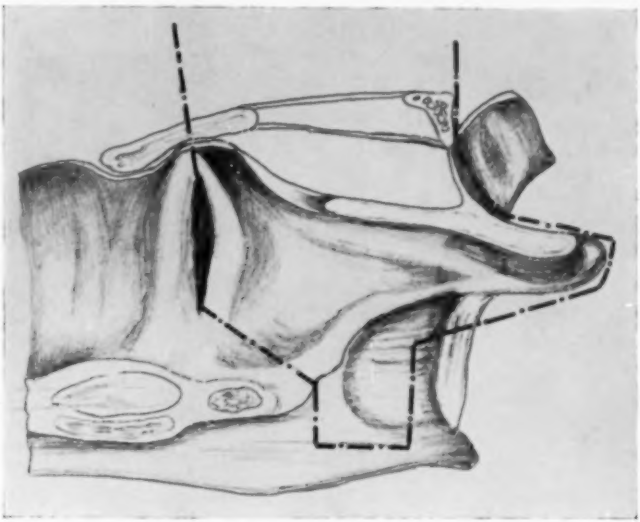

Fig. 2. Surgical Criteria. Lateral view of epiglottis and pre-epiglottic space; vertical section. The dotted line includes the area removed with a radical neck dissection.

preepiglottic space, and anatomically this space is quite separate from the true vocal cords.[13] In less advanced epiglottic carcinoma, when the lower edge of the tumor is at the region of the petiole, there may be microscopic evidence of further extension of tumor to the false cords. Extension to the anterior commissure of the true cords, ventricle, aryepiglottic fold or arytenoids is a late manifestation. In the latter instance, a bilateral neck dissection with total laryngectomy,

in my experience, has often been necessary. LeRoux-Robert[14] has demonstrated that carcinoma of the epiglottis seldom extends below the cords into the subglottic area; hence, on an embryological, anatomical and pathological basis it should be possible, in selected instances, to remove the entire lesion and preserve the function of the true vocal cords if the resulting pharyngostoma could be closed.

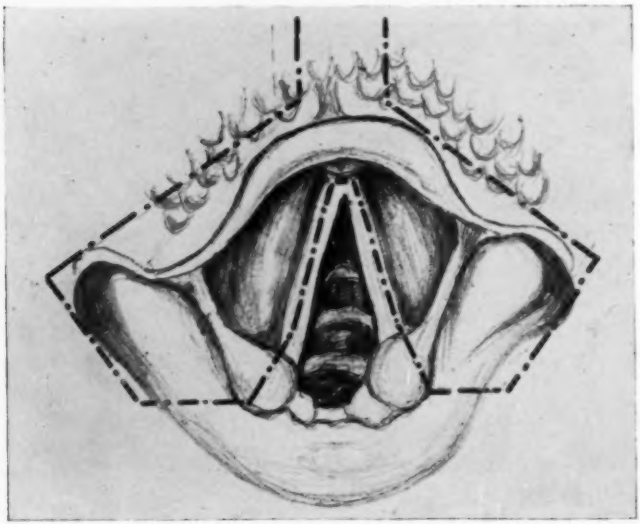

Fig. 3. Surgical Criteria. Mirror view of larynx; dotted line indicates area of excision.

Some surgeons have tried to solve the problem of surgical excision by a transhyoid pharyngotomy approach and amputating the epiglottis without horizontal transection of the thyroid cartilage. Obviously, the entire preepiglottic space cannot be removed by this method. The laryngofissure approach has the disadvantage in that the upper end of the incision will open the preepiglottic space and divide the cords at the anterior commissure.

Since metastases to the lateral cervical nodes are frequent

Fig. 4. The Incision. Note curved incision to minimize secondary contraction.

from epiglottic carcinoma, continuity *en bloc* removal of the primary lesion and the cervical nodes must be done as a one-stage procedure if one is to maintain good oncologic principles. Such an operation would create a large pharyngeal opening and would necessitate some method for closure. In South America a secondary operation has been found necessary.[8,9] It is my purpose to demonstrate that primary closure

Fig. 5. Radical neck dissection completed. Note attachment to hyothyroid area; sternohyoid and hyothyroid muscles are severed and hyoid bone is divided in midline.

of this defect can be accomplished in one operation with restoration of the deglutition and cordal function.

SURGICAL CRITERIA.

1. The tumor involving the epiglottis should be limited entirely to the cartilaginous laryngeal surface, preferably more towards the free surface (see Figs. 2, 3).

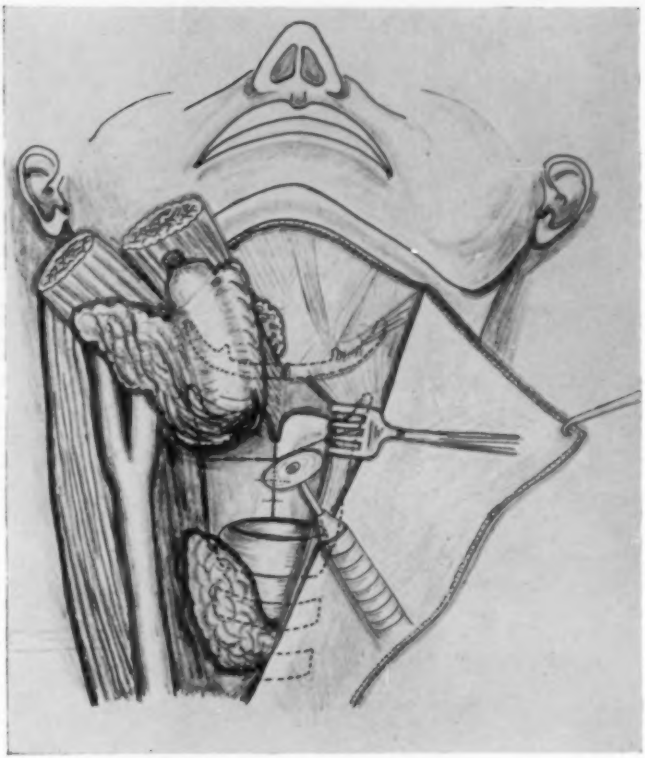

Fig. 6. Thyroid cartilage is divided transversely one-third the distance from thyroid notch to the inferior border.

2. The lower border of the tumor should not extend below the petiole. It is better if there is a greater margin of safety between the lower border of the tumor and the anterior commissure of the true vocal cords.

3. Lateral infiltration of the arytenoepiglottic folds and arytenoid edema must be absent.

4. The base of the tongue should be clear of tumor—(when

Fig. 7. The pharynx is entered in the region of the vallecula and lateral glosso-epiglottic fold.

the tip of the epiglottis on the glossal surface shows some tumor, a section of the base of the tongue is removed).

5. The true vocal cords should be free of tumor and show no impairment of motion.

6. There should be no gross evidence of false cord invasion.

7. Because extension into the preepiglottic space occurs less

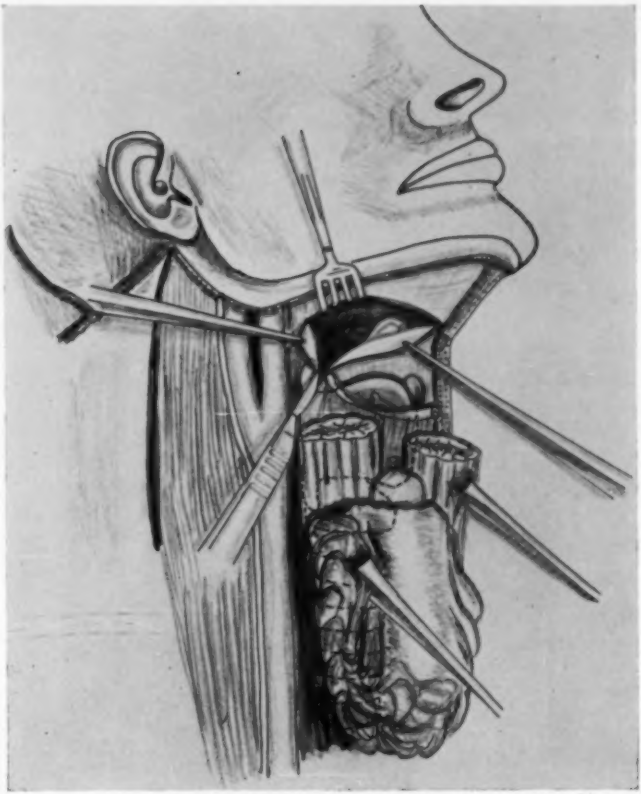

Fig. 8. The lateral laryngo-pharyngeal wall is opened inferiorly along the posterior laryngopharynx.

frequently with exophytic lesions, they are more favorable clinically than those that are infiltrative and ulcerative.

8. Clinical invasion of the hyothyroid membrane should be absent.

9. If nodes are not palpable, a neck dissection is done on the side on which the tumor seems to show the greatest extent.

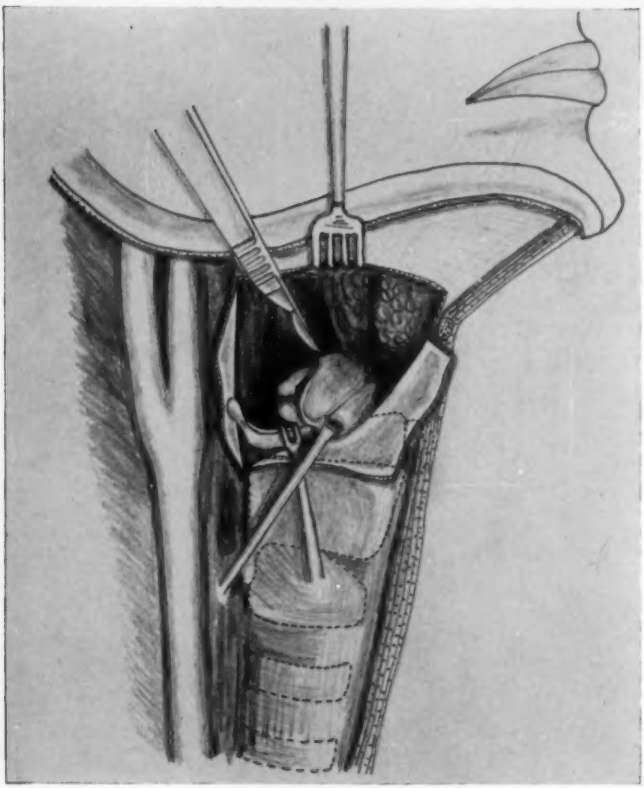

Fig. 9. The base of the tongue is separated from the epiglottis across the midline.

10. Bilateral palpable nodes warrant a bilateral neck dissection.

11. Laryngograms using Dionisil as a contrast media are of great value.[15]

TECHNIQUE.

A preliminary tracheotomy is done under local anesthesia

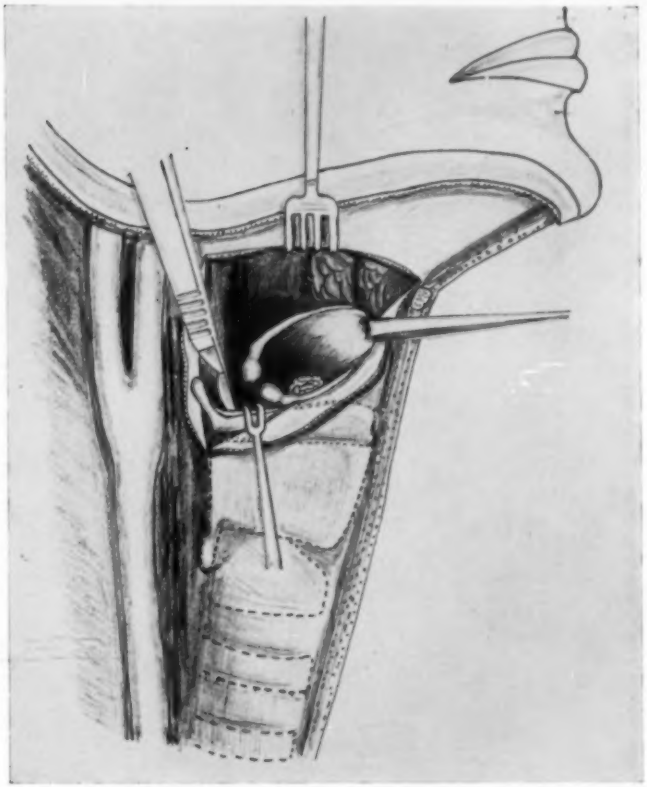

Fig. 10. Dissection inferiorly, and posterior to the superior cornu of the thyroid cartilage towards the horizontally transected thyroid cartilage.

and endotracheal anesthesia is started. A neck dissection is carried out in the classical fashion described by many authors over the past five decades.[8,16-20] The classical incision that has been modified and described by Huffman and Lierle has been used in this operation (see Fig. 4). The neck dissection in this instance differs from the usual procedure in the following aspects: No attempt is made to preserve the spinal accessory nerve whether or not nodes are palpable. The

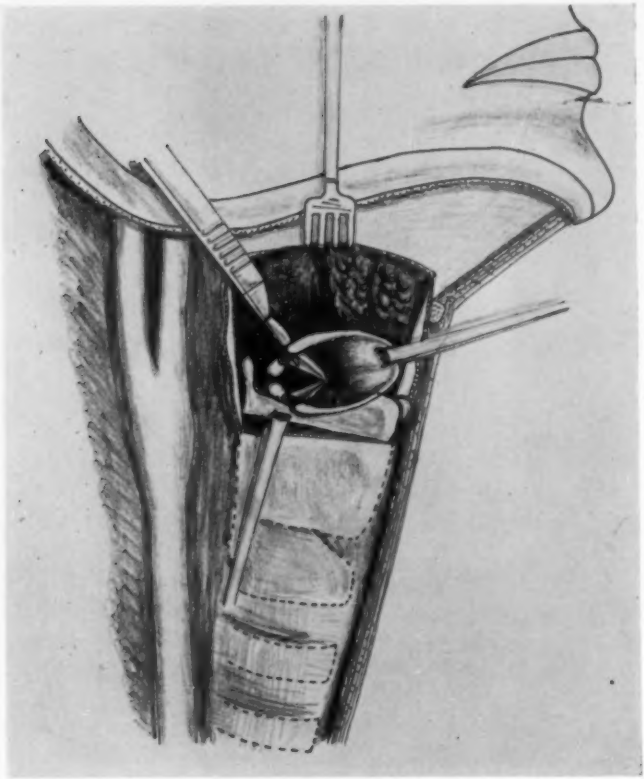

Fig. 11. The aryepiglottic folds are sectioned close to the arytenoid, and carried anteriorly into the horizontally transected thyroid cartilage; this separates the false cord-ventricle from the true vocal cords.

sternothyroid and sternohyoid muscles are transected at the lower level of the thyroid cartilage on the side of the tumor to the midline. A complete block neck dissection is performed, but the specimen is left attached to the lateral thyrohyoid area. The thyroid lobe on the side of the neck dissection is removed, and great care is given to preserve the recurrent laryngeal nerve.

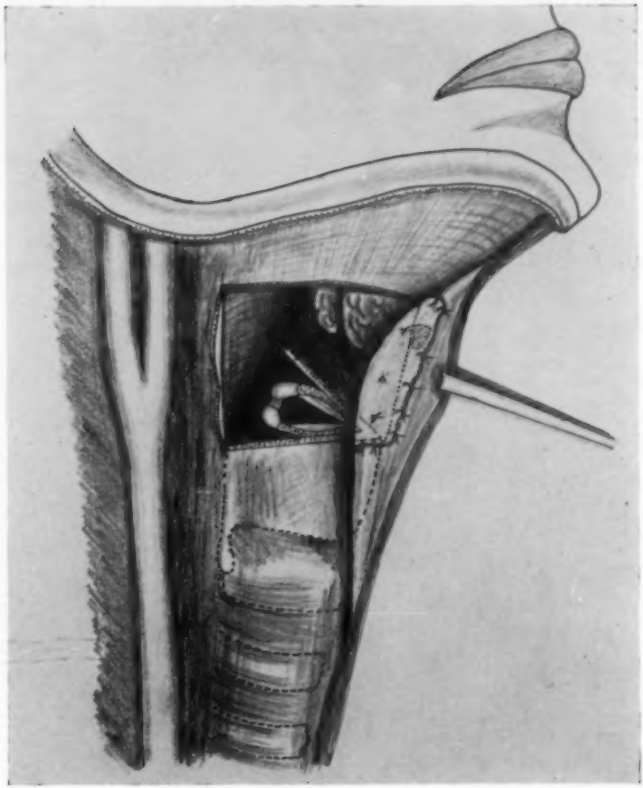

Fig. 12. A thick split thickness skin graft is sutured on a muscle rotation flap.

The suprahyoid muscles are separated from the hyoid bone on the side of the neck dissection to the midline, and the hyoid bone is divided in the midline and grasped with a tenaculum to be held inferiorly for a landmark (see Fig. 5). It is not separated from the specimen.

The thyroid cartilage is transectioned with a Stryker saw at a level one-third of the distance between the thyroid notch and the inferior border (see Fig. 6).

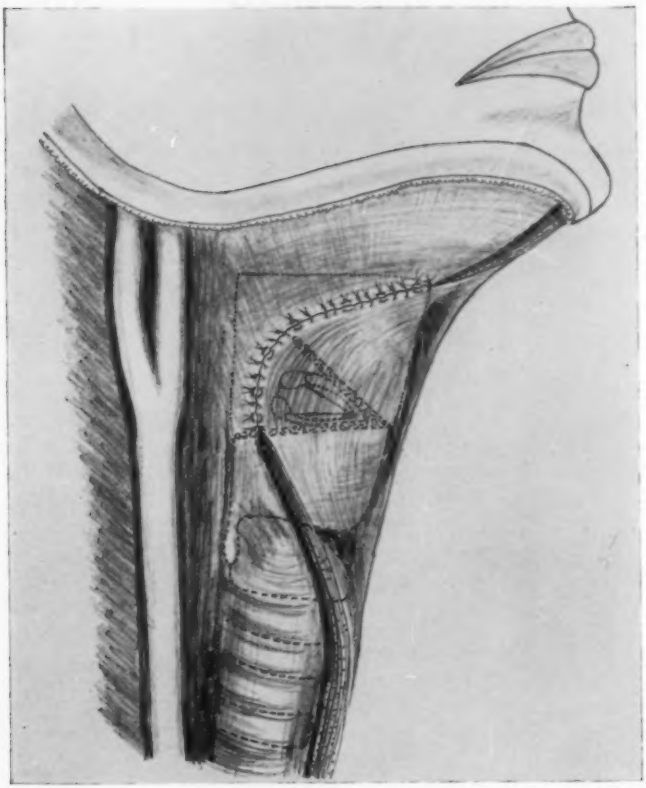

Fig. 13. Laryngopharynx approximated with two layers of interrupted silk.

On the side of the neck dissection, the sternothyroid and sternohyoid muscles are divided transversely from the midline toward the side of the neck dissection.

A pharyngostoma is created by entering the pharynx superior and lateral to the vallecula (in the region where the suprahyoid muscles were separated from the hyoid (see Fig. 7). With sharp dissection the entire lateral laryngopharyngeal wall is separated from the posterior wall, and the in-

cision is carried posteriorly to the superior segment of the horizontally transected thyroid cartilage. For further mobilization, the base of the tongue is separated from the epiglottis across the midline (see Figs. 8, 9, 10).

The ipsilateral aryepiglottic fold is now sectioned close to the arytenoid cartilage. Care should be taken not to expose the arytenoid cartilage. The ipsilateral false cord is separated from the true cord, and at the same time further separation is carried anteriorly and laterally into the previously transected portion of the thyroid cartilage. Dissection is then

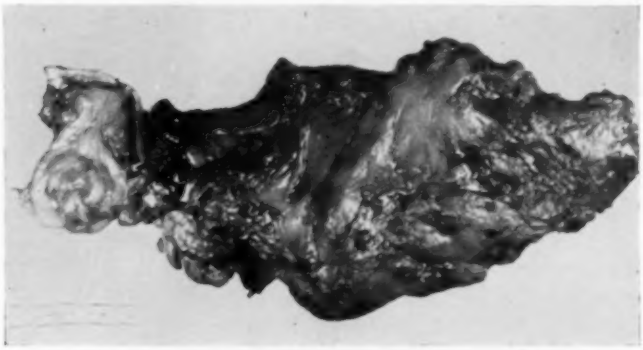

Fig. 14-A. Specimen removed.

carried towards the anterior commissure of the true vocal cords separating it from the ventricle, false cord and epiglottis. As dissection is carried across to the opposite side it will include a segment of the opposite transected thyroid cartilage, but less lateral laryngopharyngeal wall is sacrificed. The contralateral false cord is divided from the true cord and final separation of the contralateral aryepiglottic fold close to the arytenoid is the last step (see Fig. 11). In this manner the entire preepiglottic space together with the epiglottis, false cords, aryepiglottic folds and most of the ventricle (except its inferior surface represented by the superior surface of the true cord) is removed.

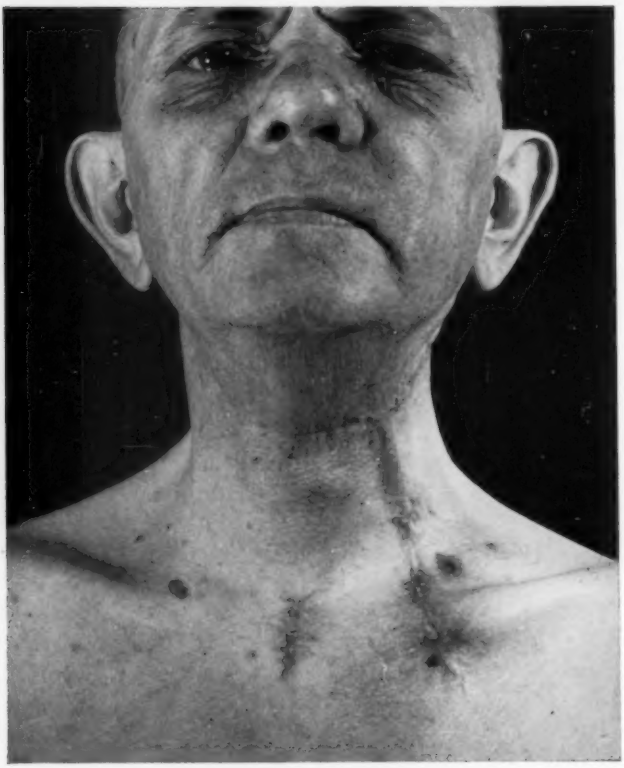

Fig. 14-B. Post-operative result.

A nasal feeding tube is passed into the stomach. A large muscle rotation flap is now created (see Fig. 12). The opposite sternothyroid, thyrohyoid muscle is dissected free from the larynx and trachea and from the hyoid bone. The flap can now be rotated, and a thick split thickness skin graft is sutured to the under surface of the muscle. The muscle flap and skin graft close off the pharyngeal defect by approximating it with the constrictor muscles. Uusually two or three layers of reinforcing sutures will be necessary (see Fig. 13).

A Penrose drain is placed into the lateral neck area, and the skin is closed by approximation of the subcutaneous tissues with silk and interrupted silk for the skin. A tracheotomy tube is inserted following removal of the endotracheal tube.

In the event that a bilateral neck dissection is necessary, then there are no strap muscles to rotate since they are removed with the transected thyroid cartilage. In this case the entire tongue is mobilized and the laryngopharynx is obliter-

Fig. 15-A. Specimen removed; bilateral neck dissection.

ated by suturing the base of the tongue to the remaining larynx. In the postoperative period, antibiotics and feeding by nasal tube is started.

RESTITUTION OF DEGLUTITION.

It is obvious that since both superior laryngeal nerves are sectioned by this supraglottic approach, feeding difficulties will develop.

After a period of ten days to two weeks, the patient is allowed to take food by mouth. The patient must relearn

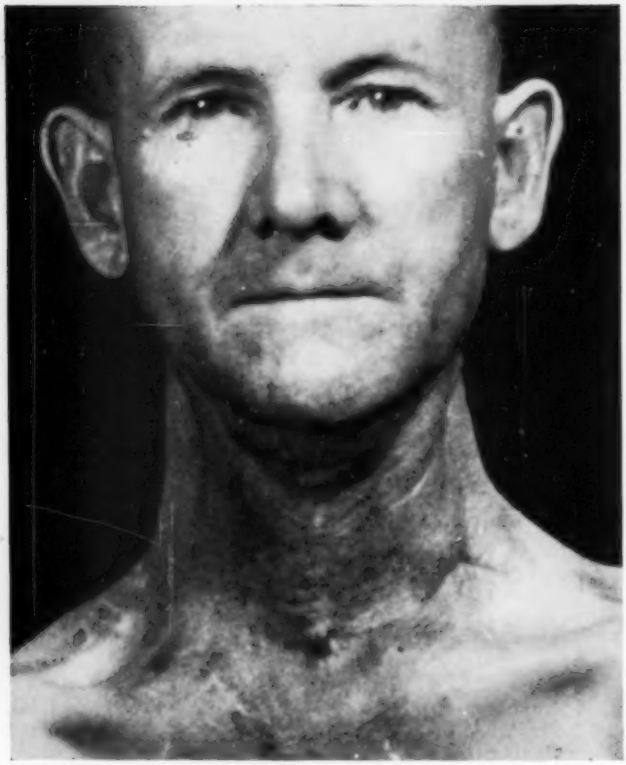

Fig. 15-B. Postoperative result.

deglutition. At first it is a conscious effort on the part of the patient, but later swallowing becomes a reflex act. Thin cereal is tolerated more easily than water until the patient becomes accustomed to the sensation of the passage of food. In the initial attempts at swallowing, the tracheotomy tube is closed with a cork, and the patient is told to swallow and breathe out utilizing the larynx to clear the mucous and food from the true vocal cords. Usually for one small amount of food this process of clearing the throat with swallowing must be repeated several times to swallow a bolus effectively. At

first some material may be aspirated into the trachea, but after several days of effort all of our patients were able to overcome this handicap. Once effective deglutition is established, the tracheotomy opening may be closed. All of our patients have a good voice immediately.

RESULTS.

Fifteen patients with epiglottic carcinoma have been operated upon with unilateral neck dissection and one with a simultaneous bilateral neck dissection (see Figs. 14, 15). In four other patients with lateral laryngopharyngeal wall cancer, the transhyoid approach without removal of any portion of the larynx entailed closure of the pharynx by the rotation of a muscle flap and skin graft. Experiences in the entire group thus far extends to a three-and-one-half year followup period. No local recurrences have resulted in the remaining larynx to date. Fixation of vocal cord occurred in one instance where the arytenoid cartilage was exposed. One patient died of a coronary occlusion one year after surgery, and autopsy showed no evidence of carcinoma. Seven of fifteen patients had positive nodes confirmed pathologically. The high frequency of metastatic nodes from epiglottic carcinoma is suggested in this small series.

Secondary neck dissection has been necessary in one instance. In this patient a node became palpable six months after the first operation.

SUMMARY AND CONCLUSION.

Fifteen patients with carcinoma of the epiglottis have been operated upon, using a technique for *en bloc* removal of the primary lesion, together with a radical neck dissection.

Reconstruction of the laryngopharynx by a muscle flap and skin graft has been effective in the early rehabilitation of the patient.

Preservation of the function of the true vocal cord is possible. The patients can have not only a good voice and a normal airway, but also are able to swallow normally without

the protective function of the epiglottis, aryepiglottic folds or false cords.*

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PSEUDO-VERTIGO FROM TRUE CHOLESTEATOMA OF THE SKULL.*†

SHIRLEY HAROLD BARON, M.D.,

San Francisco, Calif.

True epithelial cholesteatomata originating in the cranial chamber elsewhere than in the middle ear are uncommon. They represent only a fraction of one per cent of intracranial neoplasms as a whole,[1] and are seldom, if ever, thought of as a cause of dizziness. An exception to the rule is the case to be reported in which skull X-rays revealed a large cholesteatoma of the left occipital and temporal bones. This tumor, though *not* involving the middle or inner ear, proved to be the cause of the patient's dizziness.

CASE REPORT.

Mrs. J., age 43, the wife of a physician, was seen first in June, 1953, for an acute bilateral, suppurative, maxillary sinusitis. It was not until three years later that she complained of dizziness. In the interim she was seen many times.

In February, 1954, she complained of a dullness and "popping" in her right ear following an airplane trip. In March, 1954, she complained of a peculiar sensation, a dullness in both ears. An audiogram done then showed normal hearing. There was no disease noted excepting boggy nasal turbinates suggesting a vasomotor rhinitis. In November, 1954, the patient complained of discomfort in the left side of her face with some pain referred to the front of her left ear and a "filling up" in both ears. A week later the ear sensation had disappeared, but there was still a feeling of "something being in the left side of the nose and face." This eventually disappeared.

In June, 1955, the patient complained again of a feeling of fullness in both ears with intermittent "popping" and a small tender area back of her left mastoid. This tenderness was thought to be due to an inflamed lymph node. (The patient had some scaling of her scalp in this region).

On August 2, 1955, she still complained of intermittent "popping" and fullness in her ears. Examination with a nasopharyngoscope showed a small amount of lymphoid tissue above each torus and in the vault of the nasopharynx. X-ray therapy to the nasopharynx was suggested and then administered by a roentgenologist between August 5 and August 22,

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*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Inc., Beverly Hills, Calif., Jan. 18, 1958.

†From the Department of Surgery, Division of Otolaryngology, Stanford University Medical School, and the Department of Otolaryngology, Mount Zion Hospital.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 4, 1958.

1955; (each nasopharyngeal area was treated through a lateral portal 6x7 cms.; each portal received a total of 600 *r* in air). There was temporary relief of symptoms following the X-ray therapy until December 14, 1955, when she again complained of the "popping" and fullness in the right ear, which she felt was worse than ever. Examination with a nasopharyngoscope showed the central adenoid mass to be about the same.

Fig. 1. True cholesteatoma (epidermoid) of the left occipital and temporal bones with invasion of some of the mastoid cells but without involvement of the middle or inner ear. Approximate measurement, 3.5x4.5 cms. Lateral view.

Various medications, including antihistamine and vasoconstrictor drugs, were helpful only at times.

On March 5, 1956, the patient complained of dizziness which had been present for two or three weeks. She found it difficult to describe this dizziness. It was not a whirling sensation, but an unsureness or a lightheadedness akin to giddiness. It had come on without warning and did not seem to be related to her ear symptoms of "popping" or fullness. It was worse when she looked up, but could be relieved by bending her head down or by chewing gum.

Fig. 2. Same as Fig. 1, sub-mento-vertical view.

Examination showed no change in the appearance of the nasopharynx. The eardrum membranes were normal. The audiogram showed a slight loss in hearing for low tones in both ears (15 db. at 250 and 10 db. at 500 cycles per second). A cold caloric (5 cc. ice water) test for vestibular function showed normal responses for both ears. A neurological examination revealed no abnormality.

The skull was X-rayed. The films revealed an oval zone of diminished density involving the left occipital and temporal bones encroaching upon some of the pneumatized portion of the mastoid process but not involving the middle or inner ear. This defect measured about 3.5x4.5 cms.

(see Figs. 1, 2). The lesion was considered to be a true cholesteatoma or epidermoid. It was felt that it should be removed surgically.

Surgery was done on April 2, 1956, with Dr. O. W. Jones, a neurosurgeon, who used a craniotomy approach over the site of the cholesteatoma. A small horseshoe-shaped incision was employed, the anterior limb starting at the tip of the mastoid process, curving upward, then backward and down. When the pericranium was reflected caudally, grumous, pearly, gray material, characteristic of a cholesteatoma was

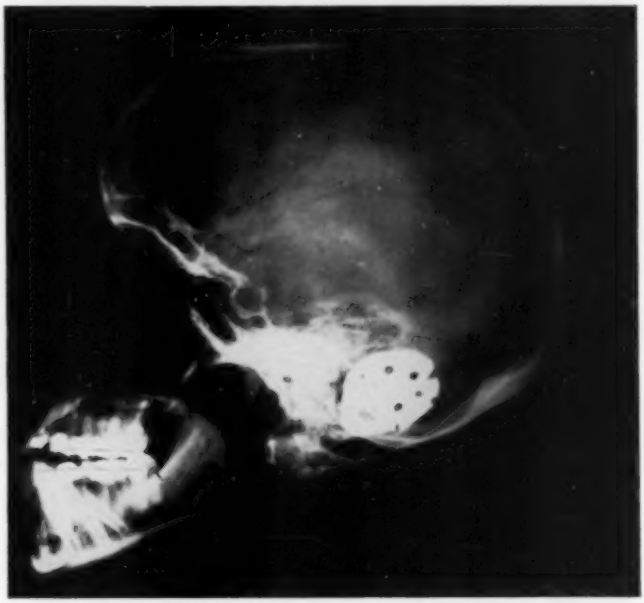

Fig. 3. Tantalum plate covering the defect in the skull resulting from surgical removal of the cholesteatoma. Lateral view.

found posterior to the tip of the mastoid process. Further exposure was obtained by removing some of the outer table of the skull toward and including the occipital bone. It was then possible to remove the sac of the cholesteatoma from the dura which covered the left cerebellar hemisphere and the lateral sinus. The dura was compressible and transparent, and about one-half inch below its surface the partially atrophic cerebellum could be seen. There was fluid between the dura and the cerebellum. The attachment of the tentorium had been displaced mesially.

After Dr. Jones removed the sac from the dura, I removed that portion of the sac wall which had eroded the mastoid cells. This was done with magnified vision and required removal of some of the underlying mastoid

Fig. 4. Same as Fig. 3, sub-mento-vertical view.

cells for complete elimination of the matrix. When these cells were removed, the mastoid antrum came into view. Its lining membrane appeared healthy and uninvolved.

Gelfoam was placed into the mastoid antrum and into the defect in the mastoid cells made at surgery and by the lesion itself. A tantalum plate was fashioned for the defect in the skull and held in place with two sutures of tanalum wire (see Figs. 3, 4). The scalp was closed, layer by layer, with sutures of nylon.

The course was uneventful. The left tympanic membrane, examined the day after surgery, showed the bluish color of a moderate hemotympanum, which disappeared within four weeks. There was a feeling of

fullness, but not of deafness. The sutures were removed on the fourth postoperative day. The patient was dismissed on the next day.

The dizziness disappeared following surgery and has not recurred to date. The patient has complained of a sensation of an echo in her left ear but feels that her hearing is normal. She still experiences "popping" in both ears. An audiogram done on June 14, 1957, revealed a slight loss of hearing as compared with that of February, 1954, but the slight loss was about equal for both ears. The readings were: for the right ear, 250-20, 500-10, 1000-0, 2000-5, 4000-10, 8000-15; for the left ear, 250-25, 500-10, 1000-minus 5, 2000-10, 4000-10, 8000-5.

COMMENT.

The discovery of such a large lesion of the skull in this patient, the wife of a physician, after she had been under my care for three years, was a bit embarrassing for, considering its size, the cholesteatoma must have been present all of those three years, probably longer; yet, in retrospect, there was nothing that suggested an indication for X-rays of the skull until the symptom of dizziness appeared.

The use of nasopharyngeal irradiation, worthless to this patient, merits criticism. This is self-criticism and criticism of others who have developed the habit of resorting to irradiation therapy as a crutch for the patient with tubal symptoms when no other treatment seems available. Irradiation therapy in patients with more defined tubal symptoms has proven disappointing in my experience,[2] so certainly there was little excuse for my having suggested it here.

The sensation of an echo in the left ear is difficult to explain. The alteration in the mastoid air cell system could hardly be considered a cause, in view of the fact that an echo sensation does not exist in those patients who have had mastoidectomies which result in a much greater alteration in the mastoid anatomy.

It is unlikely that the intermittent ear "popping" is related to the skull cholesteatoma. It is probably the result of the vasomotor instability of this patient.

DISCUSSION.

Since there occasionally exists some confusion in the terminology of the classification of cholesteatomata, it might be worth while to review Wittmaack's[3,4] classification, which is generally used. Sometimes the terms "true cholesteatoma" and "primary cholesteatoma" are used interchangeably, but incorrectly so. Recently Day[5] titled a paper, "A Primary Cholesteatoma of the Middle Ear," but in the text of his article he referred to the lesion as a "cholesteatoma verum" or a true congenital cholesteatoma. The incorrect use of "primary" in this instance was probably an oversight.

A cholesteatoma is a tumor which is surrounded by a thin shell of epidermis and connective tissue in which is accumulated desquamated horny epithelium. Wittmaack classified

cholesteatomas into two main types: *1.* true cholesteatoma, and *2.* pseudocholesteatoma. The pseudocholesteatomas are divided into: *a.* the primary, or genuine, type, and *b.* the secondary type.

True Cholesteatoma: This tumor is considered to be derived from an embryonic rest and consists of concentric polygonal lamella, composed of epidermoid cells which are devoid of nuclei. Between these lamellas are found cholesterol crystals in greater or lesser quantity. Surrounding the mass is a membrane, the cholesteatoma matrix, which has an outer layer of connective tissue attached to bone, and an inner epidermal layer. These are referred to in Bancroft[6] as "epidermoids." He states, "Epidermoids, sometimes loosely called 'cholesteatomas,' occasionally arise in the diploe of the skull. The lesions are congenital and are really cysts lined with squamous epithelium and entirely filled with flaky white epithelial debris."

There is no recognizable difference pathologically between the "true" and the "pseudo" cholesteatoma.

The true cholesteatoma, also known as pearly tumor, is found in the skull, not usually associated with the ear.

Cushing,[1] in 1922, in a very excellent article, associated the names of Cruveilhier, Johannes Mueller and Virchow with the true cranial or intracranial cholesteatoma, and credits Cruveilhier with coining the name "pearly tumor."

Cushing stated, "The cholesteatomata remote from the temporal bone are unusual tumors, and certain types of them excessively so. They are so uncommon, indeed, and excite so much interest when disclosed unexpectedly postmortem, or more rarely in the course of an operation on the brain, that they are prone to find their way into medical literature. So far as I am aware, the condition has never been diagnosed except at autopsy or operation. Certain of these lesions, however, despite their obscurity, should be quite capable of recognition because of the characteristic changes in the bone which the X-ray may disclose."

Cushing reported an interesting case of a very large choles-

teatoma of the temporal bone which had not been recognized previously in spite of the fact that the X-ray showed a defect in the skull three years earlier.

The more common situations of a true cholesteatoma are: *1.* in and about the temporal bone; *2.* in the leptomeninges of the cerebral base; *3.* in the cerebral ventricles, these last being endothelial tumors; and *4.* in the bones of the skull at places remote from the middle ear, such as the occipital, parietal or frontal bone. The latter are of the epidermoid variety alone and seem to originate between the two tables of the skull, the inner table being the first one to become ballooned out and absorbed as the growth enlarges. Such a tumor is represented by the lesion found in the case reported.

Cushing ventured the opinion that true cholesteatoma rather than pseudocholesteatoma may be the rule in the middle ear cholesteatomas, and that the cholesteatoma is responsible for the otitis media rather than the reverse. Except for the primary type of pseudocholesteatoma, it is unlikely that any otologist would support Cushing's view.

Pseudocholesteatoma: The primary, or genuine type is thought to come from a spontaneous perforation of Shrapnell's membrane. The perforation itself is the result of marked epitympanal absorption of air from a shutting of the tubotympanic space by adhesive bands that are thought to occur in the course of otitis neonatorum. From the margins of the perforation epithelium invades the attic of the tympanum (epitympanum) and the antrum of the mastoid process and, with the accumulation of the trapped products of desquamation, the pseudocholesteatoma gradually enlarges, causing pressure rarefaction of the underlying bone. This type usually is seen only when secondary suppuration occurs, or when complications arising from increased intracranial pressure cause death. They have been discovered during life as the result of a search for the cause of progressive deafness in certain patients.

The secondary type of pseudocholesteatoma is the sequel of a chronic otitis media in which the epithelium from the external auditory canal, or from the margin of the tympanic

membrane, invades the middle ear or the epitympanum and the antrum of the mastoid process. It progressively increases in size in the pattern described for the primary type of pseudocholesteatoma.

SUMMARY.

A report of a patient having had a form of dizziness caused by a true cholesteatoma of the skull has been presented. This tumor was situated in the left occipital and temporal bones with invasion of some of the mastoid cells but without involvement of the middle or internal ear. The origin of this cholesteatoma was probably in the diploe of the occipital bone.

This patient's dizziness, a pseudo-vertigo, was cured by the surgical removal of the cholesteatoma. The surgical technique has been described with the aid of slides of photographs in color taken during the surgery.

The dizziness undoubtedly was caused by pressure of the cholesteatoma upon the left hemisphere of the cerebellum, which evidenced some atrophy (as seen through the transparent dura) at the time of surgery. Apparently the surgical relief of this pressure permitted the return of function.

CONCLUSION.

True cholesteatoma of the skull without involvement of the middle or inner ear must be added to the list of possible causes of the varied forms of dizziness.

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516 Sutter Street.

REPORT OF A RECENT STUDY ON DIHYDROSTREPTOMYCIN OTOTOXICITY.*†

ARAM GLORIG, M.D.,

Los Angeles, Calif.

INTRODUCTION.

The ototoxicity of streptomycin (SM) and dihydrostreptomycin (DHSM) has been recognized since 1946. Soon after streptomycin first became available for therapeutic purposes untoward effects centering mainly around the vestibular system were noticed. Attention was directed towards these effects by patients' subjective complaints of vertigo, loss of visual focus, imbalance, slight sensations of nausea and even spontaneous nystagmus. On the strength of these complaints and at the suggestion of Dr. Edmund P. Fowler, Jr., the author conducted a study designed to determine the extent and location of the lesion apparently produced by streptomycin. The results of the study were reported in 1947 in the *Annals of Otology, Rhinology and Laryngology.*

Shortly after the completion of this first study, about 1948 or 1949, there appeared on the market a modification of streptomycin called dihydrostreptomycin. When dihydrostreptomycin was used as a therapeutic agent, complaints related to vestibular pathology were less numerous. Dihydrostreptomycin seemed to be less toxic than streptomycin. We were soon to discover, however, that the one set of symptoms were replaced with another. Instead of showing vestibular symptoms, numerous patients began to show evidence of hearing loss. This was indeed a complete change. It was somewhat of a surprise and, at first, quite unbelievable. Some of the patients began to have difficulty communicating when spoken

*Read at the meeting of the Western Section, American Laryngological Society, Inc., Beverly Hills, Calif., January 18, 1958.

†Research Center, Subcommittee on Noise in Industry, Los Angeles, Calif.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 4, 1958.

to in a loud voice, however, and it became apparent that further research to determine the ototoxic effects of dihydrostreptomycin was needed. With the cooperation of the tuberculosis department at the Gallenger hospital and the medical division of George Washington University in Washington, D. C., the author conducted a study to determine the amount of hearing loss produced in patients treated with dihydrostreptomycin. This study was reported in the *Annals of Otology, Rhinology and Laryngology* in 1951.

On the basis of the results of these two studies, the following conclusions were drawn:

1. Dosages of 1 to 2 gms. of streptomycin per day administered for periods of more than 45 to 50 days may produce complete loss of the labyrinthine function in many patients.

2. If the same regime as (1) is followed with dihydrostreptomycin, some 20 to 25 per cent of patients will sustain considerable hearing loss, particularly in the frequencies from 3000 to 8000 c.p.s.

3. Any patient receiving streptomycin and particularly dihydrostreptomycin should be tested routinely for changes in vestibular and auditory function. If dihydrostreptomycin is used, the hearing tests should be continued for at least six to nine months after treatment has stopped, because many cases developed hearing losses after treatment had been discontinued.

4. Because streptomycin affects the vestibular system primarily, it should be the drug of choice. Many patients will compensate for loss of vestibular function, but there is no satisfactory way to compensate for hearing loss.

LATER STUDIES.

A review of the recent literature revealed that the conclusions stated above are still valid. Reports by Anderson in the *Joural of Laryngology and Otology,* Lidén in *Acta Oto-laryngologica,* Cawthorne in the *British Medical Journal,* and Hawkins et al. in the *Antibiotics Annual* of 1956-57, confirm the results of studies made prior to 1953. There is

widespread published support for the belief that streptomycin is much more toxic to the vestibular apparatus than is dihydrostreptomycin, and that dihydrostreptomycin is more toxic to the auditory system than is streptomycin. In general the recent articles also confirm earlier estimates of the amount of drug necessary to produce toxic effects. Specifically, in the case of streptomycin, if 1 to 2 gms. per day are used, vestibular symptoms will usually occur in three to four weeks. If the dosage is limited to 1 gm. twice a week the appearence of symptoms is delayed considerably. The time of appearance of symptoms seems to be directly related to the total amount of drug used, and it also appears to be dependent upon the amount of time between doses.

Similar statements can be made about dihydrostreptomycin and its effect on the cochlear division of the VIIIth nerve. The hearing losses produced by dihydrostreptomycin are usually bilateral. They are primarily in the upper frequencies unless severe losses occur, in which event the speech frequencies also are affected. Most of the studies indicate that 10 to 15 per cent of patients treated with dihydrostreptomycin sustain a hearing loss after the total dose reaches 50 to 60 gms.

One point is very clearly made in most of the reports: when renal insufficiency exists, the toxic effects of the drug are increased, and more severe hearing loss results. This is to be expected, as streptomycin and dihydrostreptomycin both are secreted by way of the kidney, and renal insufficiency, therefore, raises the blood levels considerably above that found in patients with normal renal function, and causes increased ototoxicity.

Early in the course of the studies of dihydrostreptomycin and streptomycin ototoxicity, there was some disagreement about the location of the lesion. This author maintained in 1947 that because of the selectivity of the toxic effects for higher frequencies, the lesion is a peripheral one. It was, and it still is, difficult for me to see how a selective hearing loss in specific frequencies can be caused by any pathology proximal to the organ of Corti. I am happy to report that the

general conclusion now is that the lesion is undoubtedly in the end-organ, both in the case of the vestibular apparatus and the hearing mechanism.

RECENT DEVELOPMENTS.

A recent report of great interest to those of us concerned with the ototoxicity of these antibiotics stated that when pantothenic acid is added to streptomycin or to dihydrostreptomycin, treatment is accompanied by less toxicity. Several German workers, Keller et al. in particular, reported in April, 1956, that considerably less toxicity resulted in animals treated with either streptomycin or dihydrostreptomycin in combination with pantothenic acid. Several studies were conducted in the United States, notably by Hawkins in 1956-57, to check the validity of the German claim. None of these studies confirmed the German conclusions.

Much of the research into the effects of pantothenic acid combinations has been done with animals, particularly cats and mice. Because it appeared that pantothenic acid combinations might be less toxic in animals, the Lederle Company, in cooperation with the Department of Medicine, University of California at Los Angeles, requested that a study be conducted under my supervision to compare in human subjects the effects of the pantothenic acid combination called didrothenate (DIDRO) with the effects of regularly available dihydrostreptomycin. It was agreed that tuberculous patients would be used and that equal numbers would be treated with standard dihydrostreptomycin and didrothenate specially prepared by Lederle for experimental purposes. In this way the toxicity of the drugs could be compared under controlled conditions.

PRESENT STUDY.

A total of 149 patients started in the program, but only 100 of them are considered in this paper. The remaining 49 patients were excluded from the analysis, because they were discharged from the hospital for one reason or another before they had received enough drug to be of use in the study.

Prior to the therapy, each patient was tested in a quiet room with a standard audiometer at 500, 1000, 2000, 3000, 4000 and 6000 cycles per second. He was then tested twice a week for four to six weeks during the early part of the study. After this initial schedule was completed, each patient was tested once a week for approximately 90 to 100

TABLE I.

Number of Audiograms Per Patient.

No. Patients	No. Audiograms
100	1103
17	6-12
19	18-26
14	28-58
Least per patient	6
Most per patient	58

TABLE II.

Amount of Antibiotic Administered to Date of Report.

Patients treated with DHSM (N = 51)			Patients treated with DIDRO (N = 49)	
No. with no shift	No. with shift	No. Grams	No. with no shift	No. with shift
3	0	1- 24	3	0
12	0	25- 49	1	0
4	2	50- 74	8	1
3	1	75- 99	5	1
10	3	100-124	11	6
3	1	125-149	2	2
5	1	150-174	3	4
2	1	175-199	0	0
0	0	250-274	0	0
0	0	401	1	1
42	9		34	15

days and then once every two weeks until discharged or lost to the study for one reason or another (see Table I).

Fifty-one of the patients received 1 gm. of dihydrostreptomycin per day, and 49 received 1 gm. of didrothenate per day. Of the 51 patients receiving dihydrostreptomycin, nine showed an elevation in their auditory thresholds, and of the 49 patients receiving didrothenate, 15 showed an elevation in their auditory thresholds. Table II shows how many of the

persons in the total group exhibited an elevation in threshold, and the number of grams of dihydrostreptomycin or didrothenate the "shifters" had received. Table III shows the number of persons who acquired threshold shifts of 5 to 15 db., 20 to 30 db., and 35 to 50 db., at each of the frequencies tested.

Comparisons of the amount of threshold shift in db. at 1000 c.p.s. in the right ear show that two of the patients on

TABLE III.

Number of Patients with Threshold Shifts of 5-15 db., 20-30 db., 35-50 db.

Threshold Shift (db.)	Antibiotic	Ear	Number of patients with shift at indicated frequency (c.p.s.)					
			500	1000	2000	3000	4000	6000
5 to 15	DHSM	R	3	2	2	1	1	1
5 to 15	DHSM	L		2	2		3	1
5 to 15	DIDRO	R	5	5	3	3	4	5
5 to 15	DIDRO	L	3	4	3	4	5	6
20 to 30	DHSM	R			1			2
20 to 30	DHSM	L	1	1	2	1	1	1
20 to 30	DIDRO	R				2		4
20 to 30	DIDRO	L			1			2
35 to 50	DHSM	R			1	2	1	1
35 to 50	DHSM	L					1	1
35 to 50	DIDRO	R					1	1
35 to 50	DIDRO	L				1		1

dihydrostreptomycin acquired shifts of 5 to 15 db. and five of those on didrothenate showed a similar threshold shift. At 4000 c.p.s. in the right ear, one patient on dihydrostreptomycin and four on didrothenate showed a 5 to 15 db. threshold shift.

Threshold measurements of the left ears show that two of the patients on dihydrostreptomycin sustained from 5 to 15 db. shift at 1000 c.p.s. and four of those on didrothenate showed the same shift. Again when the shifts are measured at 4000 c.p.s. in the left ear, three of the patients on dihydrostreptomycin showed from 5 to 15 db. shift and 5 of those on didrothenate had the same shift (see Fig. 1).

At 1000 c.p.s. only one of the 100 patients acquired a shift greater than 15 db. on either drug. At 4000 c.p.s. none

showed a shift of 20 to 30 db. on either drug in the right ear. In the left ear, however, one person treated with dihydrostreptomycin showed a shift of 20 to 30 db. (see Fig. 2); also at 4000 c.p.s., two of the patients on dihydrostreptomycin showed shifts of 35 to 50 db.; one in the right ear and one in the left (see Fig. 3).

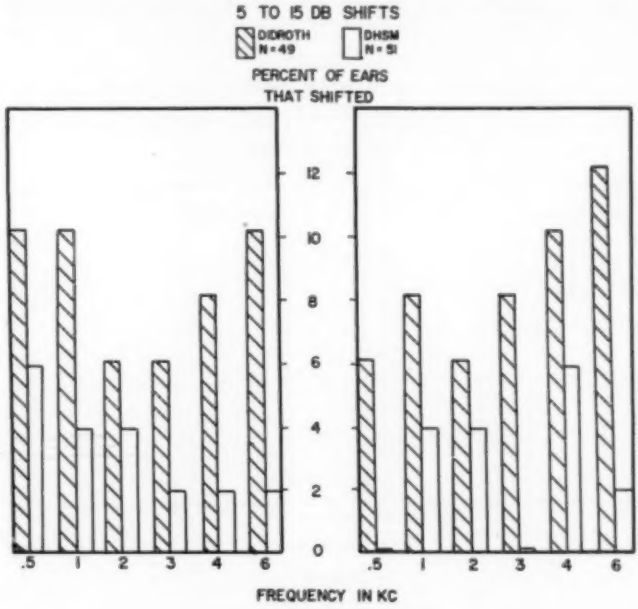


Fig. 1.

Threshold shifts were compared at 1000 and at 4000 c.p.s., because losses at 1000 c.p.s. reflect the amount of handicap for communication by speech and losses at 4000 c.p.s. represent the maximum losses usually found in the high frequencies.

There were no threshold shifts at the speech frequencies in excess of 30 db. when either antibiotic was used. On the basis of this, we can assume that no patient will sustain a significant hearing loss as a result of treatment if he has normal

hearing when the antibiotic is started. Allowing for a reasonable amount of expected variation in audiometry, some 10 db.; that is, we judge that only a relatively small per cent of the shifts are significant. Only two ears on dihydrostreptomycin and one on didrothenate showed shifts greater than 10 db. at 1000 c.p.s.

At 4000 c.p.s., we find that only three ears in the didrothenate group show more than 10 db. shift and that only five ears

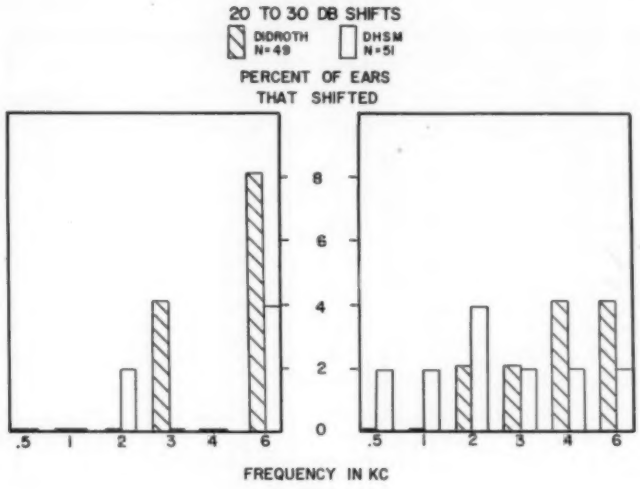


Fig. 2.

in the dihydrostreptomycin group show more than 10 db. shift. When reduced to percentage, these figures are 3 per cent and 4.9 per cent respectively.

As for losses in excess of 30 db. at 4000 c.p.s., we find that one ear shifted 40 db. when didrothenate was used and when dihydrostreptomycin was used, one ear shifted 50 db., and another 40 db. If all ears that shifted more than 10 db. at any frequency are lumped together, there are 20 in the group treated with didrothenate and 23 in the group treated with

dihydrostreptomycin. If we look for ears showing losses greater than 30 db. at any frequency, we find that there are only four with shifts of this magnitude in the group treated with didrothenate and eight in the group treated with dihydrostreptomycin (see Table III).

The significance of the magnitude of threshold shift to the patient must be evaluated from the final audiogram. In other words, is the change in hearing level enough to cause handicap? Table IV shows the number of patients whose

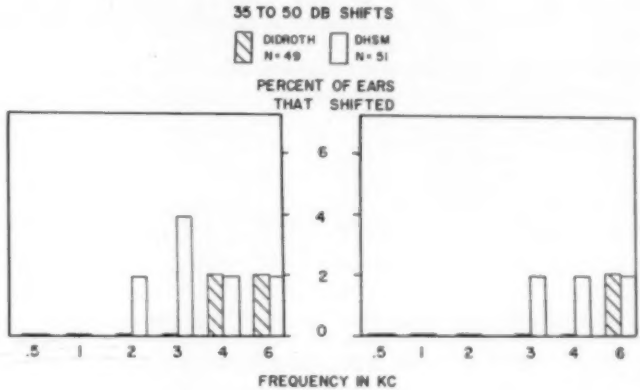


Fig. 3.

thresholds at 1000 and 4000 c.p.s. lie between 5 to 15 db., 20 to 30 db., 35 to 50 db., 55 to 70 db., and above 75 db. Only one ear shows a hearing level greater than 30 db. at 1000 c.p.s. This case was treated with dihydrostreptomycin.

To assess the amount of handicap resulting from the hearing losses acquired during this study, let us compare the beginning hearing level with the final hearing level of ears that show less than 20 db. at 1000 and 2000 c.p.s. prior to treatment and more than 20 db. at 1000 and 2000 c.p.s. at the time of this report. There is only one such case at 1000 c.p.s. in both ears; three cases, two with bilateral losses, and one

with a unilateral loss at 2000 c.p.s., in the group treated with dihydrostreptomycin. In the group treated with didrothenate, we find no cases at 1000 c.p.s. and at 2000 c.p.s., three cases with bilateral losses.

Table V shows the number of grams of antibiotic administered to cases whose hearing level on the pre-treatment

TABLE IV.

Final Threshold of Patients Showing an Elevation at 1000 and 4000 c.p.s.

Hearing Level in db.	Antibiotic Used	Right Ear		Left Ear	
		1000 c.p.s. No. Cases	4000 c.p.s. No. Cases	1000 c.p.s. No. Cases	4000 c.p.s. No. Cases
5-15	DHSM	8	4	6	2
5-15	DIDRO	13	4	12	2
20-30	DHSM	3	2	4	1
20-30	DIDRO	2		3	2
35-50	DHSM		2	1	2
35-50	DIDRO		5		5
55-70	DHSM		3		2
55-70	DIDRO		4		4
75+	DHSM				3
75+	DIDRO		2		2

TABLE V.

Amount of antibiotic used when threshold shift first appeared and total amount administered to date to patients whose thresholds at 1000 and 2000 c.p.s. were less than 20 db. pre-treatment and more than 20 db. post-treatment.

Case	DHSM No. Gms. When Shift Started	Total Dose	Case	DIDRO No. Gms. When Shift Started	Total Dose
A	125	186	A	19	150
B	42	54	B	168	258
C	130	138	C	46	84

audiogram was less than 20 db. at 1000 and 2000 c.p.s. and on the post-treatment audiogram was greater than 20 db. Note the similarity in dosages of dihydrostreptomycin and didrothenate.

Let us now consider an average of the post-treatment thresholds at 500, 1000 and 2000 c.p.s. in the better ear. If we assume than the hearing loss is significant when this average is 30 db. or more, only one patient treated with di-

hydrostreptomycin and none treated with didrothenate showed a significant hearing loss (see Table VI).

DISCUSSION.

You will no doubt remember that we started out to determine whether, in a group of 100 tuberculous patients, didrothenate (the pantothenic acid salt of dihydrostreptomycin) exhibited less ototoxicity than did dihydrostreptomycin. Let me hasten to make clear than 100 total cases in this kind of study are enough only to establish trends. We cannot draw

TABLE VI.

Number of Patients with Significant Hearing Loss After Treatment (average of 30 db. or more at 500, 1000 and 2000 c.p.s.).

DHSM	DIDRO
1	0

conclusions about the magnitude of hearing loss that will result from a given treatment. The trends are quite clearly evident, however, and I feel sure that additional studies will confirm them.

If we compare the two treatments generally on the basis of amount of shift produced, we find more patients with shifts greater than 30 db. in the dihydrostreptomycin group. Consider in more detail the magnitude of threshold shift in terms of the amount of antibiotic received by the patient. The data in Table II indicate that out of 22 patients who received less than 100 gms. of Dihydrostreptomycin, only three showed any increase in elevation of their auditory threshold. These data also show that out of 17 patients who received less than 100 gms. of dihydrostreptomycin, only two showed an increase in elevation of their auditory threshold. There were 20 patients who received between 100 and 200 gms. of dihydrostreptomycin and six showed an increase in threshold level. Sixteen patients received between 100 and 200 gms. of didrothenate and 12 showed an increase in threshold.

These comparisons seem to indicate that didrothenate is

more toxic than dihydrostreptomycin. From Table III, however, you will note that most of the threshold shifts in the didrothenate treated group are 10 db. or less. That is, they fall within our arbitrary limits of expected audiometric variability and cannot, therfore, be related to ototoxicity. Comparisons of magnitudes of shift would seem to indicate that dihydrostreptomycin is more toxic than didrothenate. The apparent difference, however, could be due to the smaller amount of dihydrostreptomycin contained in didrothenate. One gram of didrothenate contains less dihydrostreptomycin than one gram of pure dihydrostreptomycin.

When the results of this study are compared with those of my 1951 study, there appears to be a marked change in ototoxicity of dihydrostreptomycin. In spite of the larger daily dosage received by some of the patients in the earlier study, there are significant differences in the amount of threshold shift that indicate much less ototoxicity from the dihydrostreptomycin that is available today. Specifically:

1. There were no cases of profound deafness in this series.

2. Only one case treated with dihydrostreptomycin and none treated with didrothenate had a final average hearing level greater than 30 db. at 500, 1000 and 2000 c.p.s.

3. No positive conclusions can be drawn regarding vestibular effects, since no vestibular tests were performed. There were no symptoms of vestibular dysfunction, however, and we assume that there were no significant vestibular changes.

In contrast to these findings, the 1951 study included one case of profound deafness; also, out of the 40 cases treated with 1 gm. of dihydrostreptomycin per day for periods comparable to those reported in this study, 14 or 35 per cent showed losses in excess of 30 db. throughout the audiometric frequency range. About half of the impaired hearing cases showed vestibular dysfunction symtomatically and with caloric tests.

Why there is such an apparent decrease in dihydrostreptomycin ototoxicity is difficult to say. It is possible that present day manufacturing processes produce a purer and, therefore,

less toxic product; but the manufacturers have been questioned about this, and they are not aware of any changes in purity.

In view of some recent reports, we might well ask ourselves whether today's dihydrostreptomycin really is less toxic. During the past two or three years, there have been numerous oral and written reports of sudden increases in the auditory threshold of patients on small doses of dihydrostreptomycin, and combinations containing dihydrostreptomycin. These small doses vary from 1 to 10 grams. In light of our present study, it is difficult to explain these losses. The facts seem to be in order; however, the losses are real and the causal relations appear well founded. In my opinion, there can be only one explanation—individual sensitivity to dihydrostreptomycin. Such sensitivity is not restricted to the mycin preparations. We have known for many years that ototoxicity may result from quinine, aspirin, nicotine, etc. It is not unreasonable to assume that with more widespread general use of streptomycin and dihydrostreptomycin singly and in combination with broad spectrum preparations, we will be treating an increasing number of susceptible individuals.

CONCLUSIONS.

The conclusions that I wish to draw from this study are:

1. A comparison of hearing losses produced in patients treated with didrothenate and patients treated with dihydrostreptomycin definitely shows that didrothenate is not significantly less toxic than dihydrostreptomycin.

2. When the results of this study are compared with the results of previous studies, not only is the number of persons who show an elevation of threshold smaller in this study, but also the amount of threshold shift is considerably less.

3. When compared with my previous investigations the results of this study indicate that now larger total doses of these antibiotics can be administered before an elevation in the auditory threshold level appears.

4. Streptomycin is still the preferable antibiotic. It is better to lose vestibular function than to lose auditory function.

SUMMARY.

Audiometric data from tests of 100 tuberculous patients treated with dihydrostreptomycin and didrothenate (the pantothenic acid salt of dihydrostreptomycin) are presented. Fifty-one of the 100 cases received 1 gm. of dihydrostreptomycin per day and 49 received 1 gm. of didrothenate per day. Nineteen of the patients received total doses of 25 to 99 gms. of dihydrostreptomycin; 14 of them received 25 to 99 gms. of didrothenate. Twenty of these patients received 100 to 199 gms. of dihydrostreptomycin. Sixteen of them received 100 to 199 gms. of didrothenate. One received 258 gms. of didrothenate and another 401 gms. of didrothenate. One person in the dihydrostreptomycin group showed a clinically significant hearing loss. There were no clinically significant losses in the didrothenate group. The majority of threshold shifts occurred at frequencies above 2000 c.p.s.

RECOMMENDATIONS.

Five important precautions that can be used to safeguard against dihydrostreptomycin toxicity are:

1. Where either dihydrostreptomycin or streptomycin is indicated, streptomycin should be used. There is no contraindication to this procedure. Dihydrostreptomycin has never been shown to be better therapeutically.

2. When broad spectrum antibiotics are used in combination with streptomycin or dihydrostreptomycin, the combination used should contain streptomycin, not dihydrostreptomycin.

3. The manufacturers of combined antibiotics should be required to state clearly which antibiotics are contained in the combination preparations.

4. Where long term treatment with streptomycin is contemplated, vestibular and auditory tests should be given routinely.

5. When short term treatment is contemplated; that is, less than a total of 10 gms. to be administered over a period of five to ten days, the treatment should be discontinued at the first sign of vestibular or auditory toxicity. Tinnitus and slight vertigo are significant.

ACKNOWLEDGMENTS.

This study was made possible by a grant from the Lederle Company. I thank Dr. William Hewitt, Department of Medicine, U.C.L.A. School of Medicine, and Dr. Stanton Hardy of the Lederle research staff for their help on this project. I also thank Mr. Andrell Henry, the audiologist, who did most of the hearing tests.

SEVENTH INTERNATIONAL CONGRESS OF BRONCHOESOPHAGOLOGY.

The Seventh International Congress of Bronchoesophagology will be held at Kyoto (Japan) University, September 12-14, 1958, under the direction of Prof. Mituharu Goto, M.D., Otorhinolaryngological Clinic, Kyoto University Hospital, Kyoto, Japan.

A registration fee of $25.00 U.S.A. will be charged all physicians and a fee of $10.00 for wives or non-medical guests. Registered members are admitted free to banquet, receptions and sightseeing tours.

FOUR UNUSUAL CASES OF TEMPORAL BONE DISEASE.*

DAVID D. DEWEESE, M.D.,

Portland, Ore.

Review of the recent otolaryngologic literature reveals only one case report of infection of the petrous portion of the temporal bone. Lest we forget that this condition still occurs, I am presenting three cases, which I believe, emphasize the principal symptoms produced by disease in this region.

The fourth case is not one of temporal bone infection; nevertheless, it is presented because not only the area involved but also the circumstances of the involvement are unusual.

CASE REPORTS.

Case 1. A man, aged 63, was first seen on June 2, 1954, because of discharge from the right ear, double vision, and dizziness.

Symptoms began seventeen days prior to admission. First, he had pain in the right ear. Several hours later there was a bloody discharge from the ear. He was hospitalized and given terramycin and penicillin intramuscularly. This relieved the pain, but the ear continued to drain. Twelve days prior to admission he became aware of double vision and of dizziness. There was no other contributory history.

Examination of the nose, throat, nasopharynx, larynx, and left ear was normal. The right ear showed a pulsating, purulent discharge from a central perforation of the eardrum. The eardrum was thick and pink, but not bulging. The mastoid was tender.

On general physical examination, the patient was well oriented. He had complete lateral rectus paralysis in the right eye. There was partial paralysis of the VIIth nerve of a peripheral type. There was nystagmus to the left. The neck was not stiff. Temperature was 99.5 degrees.

Leukocyte count was 11,500. Roentgenogram of the mastoid showed some sclerosis with definite decalcification of the intercell walls; in addition, there was erosion of the inner table of the petrous ridge on the right side. Chest roentgenogram was normal.

He was hospitalized and given heavy doses of penicillin. The following day a radical mastoidectomy and petrous exploration were done. The mastoid portion of the temporal bone was the seat of active subacute purulent infection with cell destruction. There was no erosion of the

*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Inc., Beverly Hills, Calif., Jan. 18, 1958.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 4, 1958.

labyrinth. Healing granulations were present over one area of the sigmoid sinus. Posterior to the labyrinth there was a tract of cells. A similar tract of cells was present inferior to the labyrinth. It was possible to enter the body of the petrous superior to the labyrinth. In this area there was inflammatory tissue and soft bone, but no frank pus.

Postoperative recovery was somewhat slower than is usual after radical mastoidectomy, but except for slight fever in the afternoon, he was up, oriented, eating, and appeared well. On June 13 (ten days postoperatively) he suddenly became semi-comatose, then comatose. Fever rose to 103 degrees. There were no localizing signs.

A neurosurgical consultant followed him for four days. On the fourth day flaccid paralysis appeared in the left arm and left leg. Babinski was positive. The neck was stiff but not rigid. There was no papilledema. Leukocyte count was normal. Spinal fluid showed a mild increase in protein and 75 cells, all of which were lymphocytes.

In view of these findings, the patient was taken to the operating room for the second time (June 17). The old mastoid incision was reopened, after which the petrous was again examined. A wide examination was also made of the dura of the middle as well as the posterior cranial fossa. The dura was normal. Under the same anesthetic a neurosurgeon opened the right temporal area, and probed the temporal lobe to see if there was an abscess. None was found.

Patient's condition following operation remained poor; 48 hours later he expired.

Autopsy showed a healing mastoid-petrous wound. There was no evidence of brain abscess. Three areas of malignant metastases were found, one over the midline of the dura on the left side; the second deep in the cerebellum near the dentate nucleus; and the third in the left side near the surface of the temporal cortex. Both adrenal glands were filled with metastatic carcinoma. There was no evidence of malignancy in the mastoid or the petrous. The primary lesion was presumably a small carcinoma in the medial portion of the left lung, lying immediately behind the heart. Recheck of the chest roentgenogram taken preoperatively failed to reveal the lesion.

Case 2. A woman, aged 33, was first seen on January 26, 1956, because of persistent headache on the right side, discharge from the right ear, and low grade fever.

She stated she had had draining from the right ear since she was 18 months old. Eleven years prior to admission, after swimming, the ear became painful, and the discharge increased. A diagnosis of acute mastoiditis was made, and a "partial" mastoidectomy (whatever that is) was performed. Following this operation the ear continued to drain, and she had intermittent pain in the ear.

Three years prior to admission she began to have a low grade fever, and the discomfort in the ear increased. A radical mastoidectomy was performed at that time. The ear healed. Occasionally she had discharge from the ear for periods of 24 or 48 hours, but without pain.

One month prior to admission she had severe headache on the right side and nausea. This was followed by increased discharge from the ear, without pain. Ten days prior to admission she had difficulty in focusing the eyes, and there was some blurring of vision. The headache also recurred, and she had a low grade fever of between 99 and 100 degrees. She stated that when she was quiet she was fairly comfortable, but when she was sitting up or walking the headache was severe. She was unable to read print, except for a few minutes. She denied double

vision. Leaning or turning to the left produced nausea without vomiting. In addition to the headache she had a pain which she described as deep aching behind the right eye.

On examination, the throat, nose, and left ear were normal. The right ear showed a clean radical mastoid cavity, in which all structures were visible. The round window, the oval window with the stapes bound down, and the intratympanic course of the facial nerve were clearly visible. There was some moisture in the region of the Eustachian tube. There was a small area of exposed bone behind the labyrinth. Suctioning of the middle ear area produced slight dizziness and nystagmus. The fistula test was negative.

Blood count was normal. Roentgenograms showed the previous surgical defect. In the apex of the petrous the cells were clouded without definite bone destruction.

Neurologic examination was entirely normal except that the neurologist felt the history indicated definite meningeal irritation. An electroencephalogram was reported as abnormal because of isolated slow waves which arose in both hemispheres, but more so on the right. Extraocular muscle function, visual fields, and fundi were normal.

A diagnosis of petrositis was made, and operation was advised.

Exploration of the right temporal bone was done on January 30, 1956. The previously described mastoid cavity was clean, except for pus in one tract of cells immediately superior to the intratympanic course of the facial nerve and in another tract of cells behind the labyrinth. Exploration of the postlabyrinthine tract failed to reveal anything unusual. There was a wide space of pneumatization superior to the superior semicircular canal, making it possible to explore the petrous deeply under direct vision. The bone of the body of the petrous was granular and soft. There was granulation tissue in most of the body of the petrous to the apex. This was removed.

After operation the headache disappeared. Visual disturbance also disappeared. She was discharged eight days postoperatively. Except that the cavity was slow healing, she did well for six weeks, and then symptoms recurred. Over the next three months she had intermittent headache and intermittent blurring of vision. Then all symptoms subsided.

Ten months postoperatively she began to have fever, which slowly increased to 102 degrees. There was also recurrence of retro-ocular pain. In addition, she had some chilly sensation, but no real shaking chills. Examination at this time showed the ear to be moist. All other studies, including neurologic examination, were within normal limits.

She was carefully followed for one month. During this period the temperature ranged from 99.5 to 102 degrees. The only complaints otherwise were a feeling "as though I were being pulled over backwards," and increasing drowsiness.

Because of the fever and other symptoms, as well as the persistent discharge, the temporal bone was again explored, on November 23, 1956, 11 months after the first exploration. In the angle between the superior semicircular canal and the middle fossa dura a piece of sequestrated bone was found. The petrous body and apex were again filled with soft granular bone which had the appearance of proliferative osteomyelitis. This was removed. The inner table of the roof of the petrous was removed toward the tip, until solid bone was encountered in all directions. Although the nerve could not be identified, the internal auditory meatus must have been approached since there was twitching of the face. Fur-

ther removal of bone was discontinued. A small polyethelene tube was placed in the petrous tip, and brought out into the mastoid cavity.

The tube was slowly removed over a period of six weeks. Penicillin and chloromycetin were used continuously throughout hospitalization, and for six weeks after discharge. The ear healed primarily. Since that time the patient has been clinically well and symptom free.

Case 3. A man, aged 49, was first seen on June 28, 1955. His chief complaint was progressive hearing impairment in both ears, of three years' duration.

He stated that during examination in 1952 his attention was called to the fact that the right side of his face appeared to be weak. There was, however, no paralysis. He gave no history of tinnitus or dizziness.

Examination of the nose, throat, nasopharynx, and larynx was normal. The palate rose normally. The tongue and vocal cords moved normally. There was weakness of all branches of the VIIth nerve on the right side. The corneal reflexes were normal. The left ear was entirely normal. The right eardrum was intact. *Through the eardrum* a small white area was visible anterior to the short process of the malleus.

The audiogram was normal up to 2,000 cycles per second. There was a high tone loss of hearing in both ears, somewhat worse on the *left* side.

Roentgenograms of the mastoid, the skull, and the internal auditory meatuses were normal. Electrical tests of the right facial nerve showed equal chronaxie on both sides, but elevation of the voltage required and of rheobase on the right side.

A presumptive diagnosis of neuroma of the facial nerve was made, and exploration advised. The patient returned to his original physician, who advised strongly against any type of operation on the ear.

Seven months later, on February 7, 1956, he came back for re-examination. In this interval his face had gradually become more paralyzed. His eye was tearing. He had had some paresthesias of the right side of the tongue, and one or two momentary spells of true vertigo.

Examination showed a definite small white mass which was pushing Shrapnel's membrane out in front of the malleus. Electrical testing of the right VIIth nerve gave results identical with those obtained by testing seven months previously. A complete neurologic examination was otherwise normal.

This time the patient accepted advice for surgical exploration, but operation was not performed until June 6, 1956. The mastoid was normal, and well pneumatized. The antrum was normal. After removal of the bony bridge over the aditus, it became evident that the attic was full of a mass which appeared to be cholesteatoma. The incus and head of the malleus were removed. The horizontal semicircular canal was prominent. There was no evidence of a superior semicircular canal. The normal position of the superior semicircular canal was occupied by a large cholesteatoma, which filled the medial part of the attic. It extended underneath the intratympanic portion of the facial nerve as well as over the labyrinth and on top of the body of the petrous. An area of the inner table was eroded, and the dura was exposed for an area one-and-a-half cm. in diameter in the floor of the middle fossa medial to the labyrinth. The intratympanic course of the facial nerve had been completely exposed by the cholesteatoma. All of the cholesteatoma was removed, and the facial nerve was decompressed to the stylomastoid foramen.

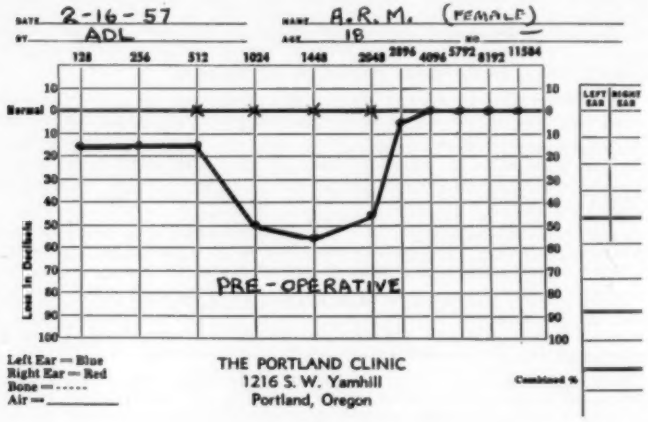
The pathologic report confirmed the clinical diagnosis of cholesteatoma.

Postoperatively the radical mastoid cavity healed normally. Because of

the facial paralysis, which became complete, electrical treatments were carried out daily. Two-and-a-half months postoperatively slight motion of the nasalus muscle was suspected. This was more definite by the end of four-and-a-half months.

The patient was not seen again until June 21, 1957, approximately one year after operation. Examination showed definite motion of all branches of the right facial nerve except the frontalis. The patient said he had noticed beginning motion in March of 1957. He also said that on occasions he had had a momentary feeling of propulsion, but no real vertigo.

Audiograms at this time showed 50 decibels loss of hearing up to 1,500 cycles per second. There was a sharp drop above that to 100 db. loss at 6,000 cycles per second.



Case 4. A girl, aged 18, was examined on February 16, 1957, because of hearing loss and low pitched buzzing tinnitus.

Seven months prior to admission, on July 21, 1956, she had been the guest passenger in the front seat of a car. An accident occurred, during which she was thrown forward and hit the left side of her head against the dashboard. There was no facial laceration or unconsciousness. The left ear bled some, but it was not treated.

She noted loss of hearing in the left ear immediately after the accident, and for one week after the accident she had a loud buzzing tinnitus. This improved somewhat, but a low pitched buzzing tinnitus persisted. There had been no vertigo at any time. She denied past ear disease, and claimed entirely normal hearing prior to the accident; however, she had never had the hearing tested.

Examination disclosed no abnormalities except for the left ear. The eardrum on this side appeared to be slightly retracted. The incudostapedial joint was visible through the drum, and appeared to be in normal position. The above audiogram of the left ear showed:

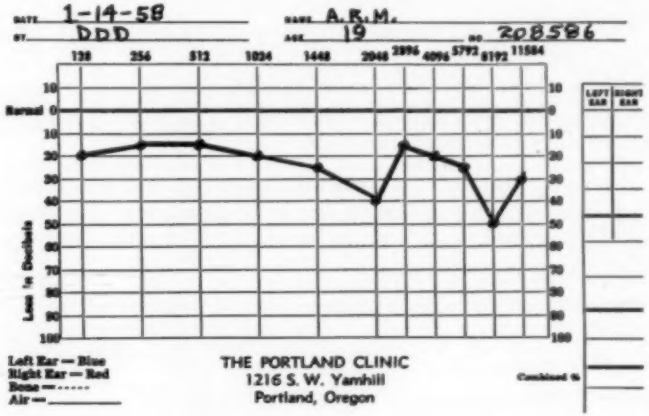
This is a pure conductive hearing loss. Inflation of the Eustachian

tube on the left side produced only a 10 decibel improvement at 1,500 cycles and a 5 db. improvement at 2,000 cycles. With both 500 and 1,000 cycle tuning forks the Weber test was constantly lateralized to the left ear, even when the right external auditory canal was occluded. The Rinné test was negative on the left side with both forks. These tests had been done previously by two other otolaryngologists.

Roentgenograms of the mastoid showed normal, well areated temporal bones on both sides.

A diagnosis of probable dislocation of the incus was made.

On March 27, 1957, eight months after the injury, exploratory operation was performed. Since it was anticipated that the incus might have to be removed, the middle ear was approached through the postaural incision. Enough mastoid cells were removed to permit easy visualization of the middle ear. There was a large zygomatic extension of air cells, which made it possible to expose the attic of the middle ear without disturbing the soft tissue of the external auditory canal or the eardrum.



Careful removal of the inner quarter of the bony external canal, down to the horizontal semicircular canal, allowed visualization of the ossicles. The incudo-malleolar joint appeared to be normal. The superior malleolar ligament was normal. The incus was completely dislocated from the stapes. Directed anteriorly, it was holding the stapes partially fixed; however, there was some mobility of the stapes. Since it was not possible to replace the incus on the head of the stapes so that it would remain, it was therefore removed. The cavity was closed.

The above audiogram of the left ear, taken on January 14, 1958, ten months postoperatively, showed:

The result is best stated in the patient's words: "I hear 100 per cent better, and I notice it mostly in conversation."

Although the final hearing result is not normal, it falls in the range of better than 25 db. in all speech tones except those of 2,000 cycles per

second. These findings would tend to confirm the stated opinion of others that serviceable hearing is possible without the ossicular chain. They also support the feeling that an intact ossicular chain is responsible for 20 to 25 decibels of hearing.

Speech reception threshold was recorded at 26-28 decibels. Tolerance to loud sound was normal. Ninety decibels of sound, pure tone, and white noise were not uncomfortable.

COMMENT.

The variation in the histories of the first three patients emphasizes the different symptoms and signs which can be produced by disease of the petrous portion of the temporal bone. These are: annoying recurrent pain, usually behind the eye; persistent aural discharge; paralysis of the VIth and VIIth nerves; meningeal irritation; vertigo.

The first patient had all symptoms and signs except retro-ocular pain. The second had all signs except VIth and VIIth nerve paralysis, but there were signs of ease of ocular muscle fatigue. The third patient showed only VIIth nerve paralysis and mild, nondiagnostic vertigo.

Roentgenograms of the petrous (Stenver's and base plates) gave valuable information in two of the patients. These are necessary additions to routine mastoid films when any deep involvement is suspected.

The case of primary cholesteatoma is interesting because it is rare. As in a similar case, reported by Dr. Kenneth Day,[1] the mastoid portion of the temporal bone was normally pneumatized.

Exploration and drainage of pus or removal of diseased bone is essential to successful recovery when the petrous is involved. It is, therefore, evident that the otolaryngologist must constantly review the anatomy of the deep temporal bone. Antibiotics alone are not the answer.

SUMMARY.

Three cases of disease of the petrous portion of the temporal bone are presented. Despite the rarity of petrositis, it points up the necessity of constant review of the anatomy of the deep temporal bone.

One case of dislocation of the incus is also reported. Surgical removal of this bone was followed by clinically useful hearing.

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1216 S. W. Yamhill Street.

INSTITUTE ON INDUSTRIAL DEAFNESS.

Colby College, Waterville, Maine, presents the Sixth Annual Institute on Industrial Deafness, August 11-20 inclusive. Please note that the course has been extended for three days in order to include engineering for noise control. Its objective will be to train physicians interested in the problem; nurses, plant engineers and others in initiating and conducting hearing conservation programs in noisy industries. The course includes basic otology, audiometry, noise measurement, ear protection and methods of noise control. Class is limited to approximately twenty participants.

The fee for the course is $250 and includes tuition, room and board. The first week will constitute a complete course for doctors and nurses, and the reduced fee for this will be $200. Application should be made to Mr. William A. Macomber, Colby College, Waterville, Maine.

EXPERIMENTAL OBSERVATIONS ON SQUAMOUS METAPLASIA OF THE TRACHEA.*†

NOBLE O. CORRELL, M.D.,
and
EDWARD J. BEATTIE, JR., M.D.,
(By Invitation),
Chicago, Ill.

INTRODUCTION.

In 1955 we began studies of the physiology of the trachea.[1] Since much of the research on respiratory epithelium has been accomplished by otolaryngologists we felt that our findings might be of interest to this group.

REVIEW OF THE LITERATURE.

More than 25 years ago there had been numerous observations on the physiology of the epithelium of the respiratory passages, by several different investigators.[3,4,9,10,11]

In 1956, Hilding published a series of four articles on cigarette smoking, bronchial carcinoma and ciliary action.[5,6,7,8] Proetz has also made numerous substantial contributions to the understanding of the physiology of respiratory epithelium.[12,13] A complete review of the above literature in this paper is not possible nor necessary.

We were interested in knowing whether the direction of beat of the cilia was inherent within the cell or due to some other factor. We also wanted to know whether an autogenous devascularized free tracheal graft would survive, and if so, how the epithelium regenerated.

In both dogs and chickens it was possible to excise two-

*Read at the meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society, Inc., Chicago, Ill., Jan. 13, 1958.

†From the Surgical Research Laboratories, Presbyterian-St. Luke's Hospital, and the University of Illinois School of Medicine, Chicago, Ill.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 10, 1958.

inch segments of the cervical trachea and anastomose the devascularized autogenous tracheal segment in a reversed direction, so that the cilia of the graft pointed caudad. Those animals and birds which survived were killed and autopsied after variable periods. The trachea, including the graft, was excised, fixed and stained for histological examination.

The graft showed ischemic degenerative changes throughout the first 12 days. During this period the only remnant of the epithelium was islands of reserve cells which were always in evidence. Approximately two weeks following the reversal of the graft, revascularization could be seen in the sub-mucosal stroma, and by three weeks the normal ciliated epithelium had completely regenerated from the surviving islands of cuboidal type reserve cells.

It was demonstrated that the cilia of the reversed tracheal graft retain their direction of beat with respect to the graft, thus propelling the overlying mucus stream caudad.

An incidental finding during these experiments was that in the dog, the circumferential tracheal anastomoses tended to stricture. Such a tendency could be discouraged by rotating the graft so that the membranous portions of the graft and trachea did not meet. This purposeful rotational mal-alignment resulted in a ring of solid cartilage at the anastomotic sites which resisted stenosis.

The chickens, having complete cartilaginous rings without a membranous portion, showed no tendency toward stricture of their tracheal anastomoses.

Another incidental finding was that the birds and animals with reversed tracheal grafts always carried a ring of mucus at the caudad anastomosis, where it was continuously being piled up by the cilia. These creatures were forced to cough at regular intervals to clear their mucus accumulations.

The proximal anastomosis was always dry and underwent squamous metaplasia. When the anastomoses were excised and the ends re-approximated, squamous metaplasia again occurred at the site of the proximal anastomosis. This find-

Fig. 1. Squamous metaplasia of proximal anastomosis of reversed autogenous tracheal graft.

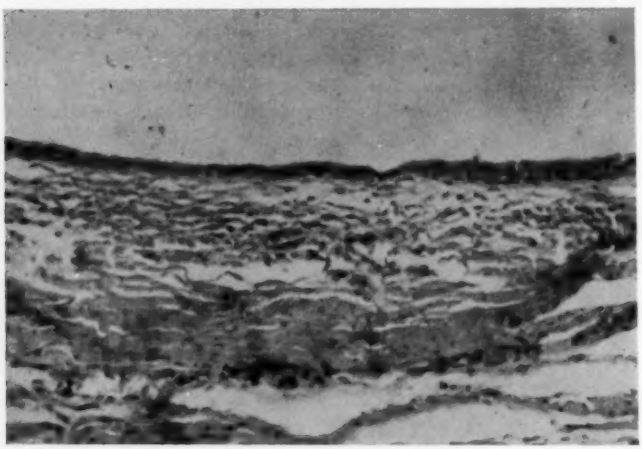

Fig. 2. Persistent squamous metaplasia of proximal anastomosis 400 days after reversal of cervical trachea in the dog.

ing was present in all of the birds and animals with reversed tracheal grafts.

The work just described has been previously reported.[1,2]

METHODS.

Four dogs were allowed to live longer than one year after their tracheal graft reversals. Two dogs were killed on their

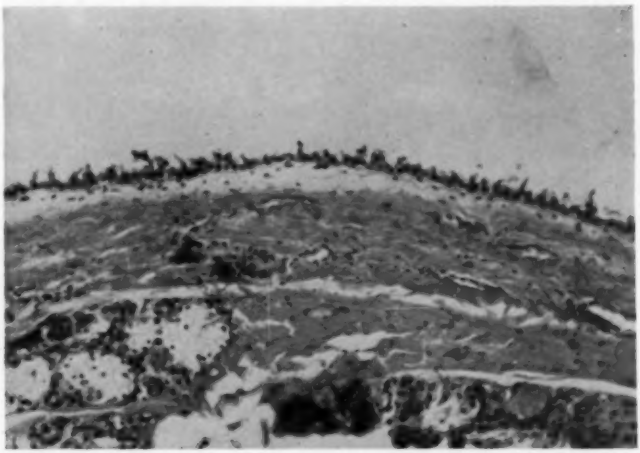

Fig. 3. Trachea caudad to reversed graft in a dog 500 days after reversal of cervical trachea and nine days after permanent tracheostomy cephalad to the graft. Note the absence of ciliated columnar cells which have presumably sloughed as the result of inflammation from retained secretions.

400th post-operative day. They were autopsied and their tracheal grafts, including the anastomoses, were prepared for histological examination. Two dogs were given permanent tracheostomies, bringing the cricoid cartilage out as a tracheal stoma on their 450th and 500th post reversal day respectively.

A fifth dog is living as a pet, with a cervical trachea reversed more than two and one-half years ago.

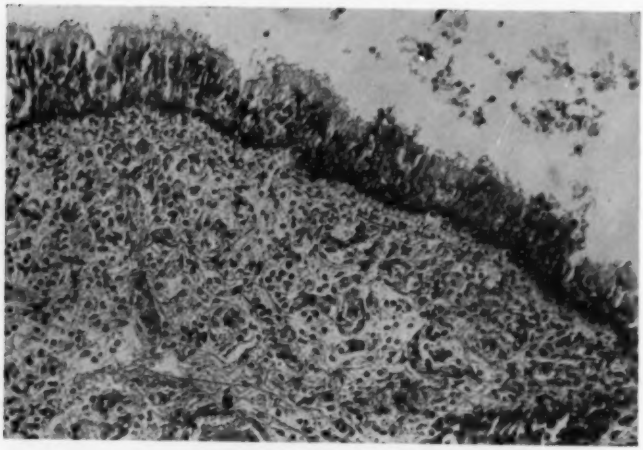

Fig. 4. The reversed graft of the same animal depicted in Fig. 3. Note the marked vascularity and inflammatory cells beneath the mucosa.

Fig. 5. A tracheal graft in the dog four months following reversal, showing a large number of mucus producing cells in the regenerated epithelium.

RESULTS.

The two dogs killed 400 days following reversal of their cervical tracheas showed heavily ciliated columnar epithelium lining the graft. There was squamous metaplasia of the proximal anastomosis which differed in no respect from the metaplastic changes found in the proximal anastomoses of all the previous specimens examined (see Figs. 1, 2).

The two dogs given permanent tracheostomies died eight and nine days later of retained secretions, with asphyxia and

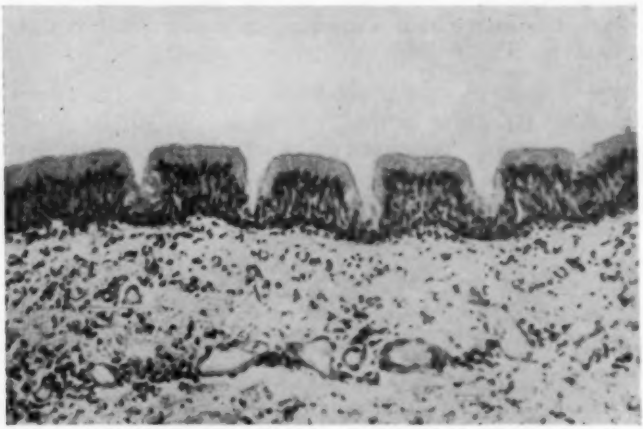

Fig. 6. Another section from the same graft as Fig. 5 showing "mucus pits."

severe tracheo-bronchitis. The epithelium of their tracheas differed from that of their grafts, in that the trachea showed uniform survival of the reserve cells only, in an orderly solid sheet (see Fig. 3), while the grafts showed more sub-mucosal vascularity and a thicker, more metaplastic and basophilic epithelium (see Fig. 4).

DISCUSSION.

Whether this heavier, better preserved epithelium is the re-

sult of a better blood supply or a distorted growth pattern brought about by the previous phase of regeneration of the graft is not known.

It does appear that the epithelium of the graft usually differs in some respects from the normal respiratory epithelium. There are more mucus producing cells in the epithelium of the grafts (see Fig. 5), and many of these cells are clumped together, forming a cluster of ciliated goblet cells located around a depression in the surface of the mucosa (see Fig. 6). These glandular, mucus producing pits, lined by cilia, are more numerous than the well-known racemose glands of the tracheo-bronchial tree. The "mucus pits" are also more shallow, the entire glandular aggregate being superficial to the basal layer of reserve cells of the epithelium.

Even the surface of normal tracheal epithelium is pockmarked by glandular orifices when examined under suitable magnification and illumination. Whether these craters represent "mucus pit" glands or openings of the racemose glands ducts is not known.

SUMMARY AND CONCLUSIONS.

1. Squamous metaplasia of the cephalad anastomosis of reversed autologous tracheal grafts persists for more than one year.

2. The mucosal lining of the grafts seems unusually well supplied with ciliated goblet cells, frequently clustered around "mucus pits."

3. The regenerated epithelium of the grafts exhibits an abnormal response to infection, the significance of which is unknown.

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UNUSUAL TUMORS OF THE TONSIL AREA.*†

LEWIS W. JORDAN, M.D.,

Portland, Ore.

INTRODUCTION.

Since disturbances of the tonsil and its surrounding structures are commonly caused by infection, it was thought that by way of contrast a discussion of five tumors in this area would be of general interest and value.

These tumors are: *1.* Osteoma of the inner surface of the mandible presenting in the left tonsil fossa; *2.* Fibro-liposarcoma arising on the dorsum of the tongue and extending into the right tonsil fossa; *3.* Mixed tumor of aberrant salivary gland in the nasopharynx invading the left posterior pillar; *4.* Malignant lymphoma of the right tonsil; *5.* Squamous cell carcinoma of the left tonsil. The first three tumors occur rather commonly in many parts of the body but are unusual in the sites to be described, while the last two neoplasms are occasionally seen only in the tonsils.

OSTEOMA OF MANDIBLE.

Case 1. Mrs. V. H., white, age 50, was referred on Nov. 29, 1954, because of an asymptomatic medial protrusion of the left tonsil which had been found during a routine physical examination. The tonsils were atrophic, and the one on the left appeared to be displaced medially by a large, firm, discrete, and fixed mass about 5 or 6 cm. in diameter coming up from the base of the tonsil fossa. The ear, nose, throat, nasopharynx, larynx, sinuses, and neck showed no essential pathology, and the general examination was not contributary. The report of a small biopsy was "chronic tonsillitis with focal ulceration."

Under general anesthesia on Dec. 13, 1954, a vertical incision made behind the anterior pillar immediately disclosed a smooth and spherical osteoma with the atrophic tonsil stretched over it. Both tonsils were removed by dissection and snare. The bony tumor was found to be attached to the inner surface of the angle of the mandible by a very broad base. By means of mastoid gouges, several large fragments of the mass were removed. The bone was so hard that the edge of one gouge was

*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Inc., Beverly Hills, Calif., Jan. 18, 1958.

†From the Department of Otolaryngology, University of Oregon Medical School, Portland, Ore.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 4, 1958.

broken. By this time it was evident that complete removal of this benign tumor would be a major procedure which was not justified by the symptoms. Accordingly, the wound was closed by suturing the anterior and posterior pillars together.

The pathology report by Dr. Warren C. Hunter was as follows: In addition to tonsils there are a number of pieces of bone, the largest measuring 2.5x1.5 cm., having roughened and somewhat cancellous surface opposite to a smooth and convex aspect. The mass cut with great difficulty and mostly appeared solid rather than cancellous.

In microsections the mass proved to be extremely dense bone, poor in bone cells and with a good many Haversian canals present. If this is other than an exostosis it can be termed an osteoma (see Fig. 1).

Fig. 1. Case 1. Photomicrograph (X160) depicting representative structure of osteoma, together with Haversian canals.

By Dec. 20 the edges of the wound had separated and the bare osteoma was widely exposed. On Jan. 21, 1955, the bony mass was still completely exposed with no evidence of progress in covering by soft tissue; however, shortly thereafter, a large superficial sequestrum was removed, which revealed healthy granulations extending over the entire osteoma. In a few days, the area was completely covered by normal mucosa.

The post-operative X-ray report of Dr. Arthur L. Hunter on Dec. 23, 1954, stated, "Radiography of the left mandible shows a huge semi-oval calcium density projecting medial to the angle of the mandible on the left side. This density measures about 5 or 6 cm. in greatest diameter and has the radiographic characteristics of a huge osteoma" (see Fig. 2).

The patient was last seen on Aug. 20, 1957, at which time the mass appeared to be unchanged in size and the patient was having no throat symptoms.

Thoma[1] described the removal of a similar tumor by an extraoral approach. The mass was so large that it interfered with talking and prevented the wearing of an upper denture. Because the osteoma was so hard it was necessary to remove part of the cortex of the mandible with the specimen. The same writer states,[2] "The development (of an osteoma) may occur in any part of the maxilla and mandible and arises from preformed bone, periosteum or retained cartilage cells from the embryonic chondroskeleton. Growth is progressive,

Fig. 2. Case 1. Osteoma of Mandible as seen by X-ray.

but slow, and the tumor is benign in character. It starts spontaneously and is believed by many to be of traumatic but noninflammatory origin. Its growth generally ceases with the termination of general skeletal development."

Uhler[3] presented the case of a man with such a large pedunculated osteoma attached to the lower first molar area that the tongue was displaced, making the intake of food difficult. The tumor was thought to be the result of a blow to the lower jaw 37 years before. Removal was accomplished with a Gigli saw.

Rosedale and Hargraves[4] reported the case of a colored man with an osteoma of the inner surface of the mandible extending from the left lateral incisor tooth as far posteriorly as the posterior pillar of the tonsil and extending vertically from the superior surface of the mandible to the inferior surface. Because of difficulty in swallowing and speaking it was removed intraorally.

True osteomas occur rather frequently in the frontal sinus. Ewing[5] states that these arise from fragments of cartilage connected with the ethmoid. Osseous change of the maxilla and mandible often is associated with dental pathology and the occurrence of an exostosis is rather common. At times, bone is combined with cartilaginous, fibrous, or other types of tissue in a neoplasm. The torus palatinus and torus mandibularis are common and usually cause difficulty only in the wearing of dentures.

FIBRO-LIPOSARCOMA OF THE TONGUE.

Case 2. Mr. M. R., white, age 45, sought treatment on Nov. 4, 1944, because of the sensation of a foreign body in his throat. He stated that in 1923 (age 24 yrs.) a tumor had been removed from the posterior part of his tongue. The record of this procedure was not available. Later, he was treated by the late Dr. Frank B. Kistner of Portland, Ore., whose record was as follows: "This patient was seen in 1927 (age 28 yrs.) at which time a large mass was found attached to the lower outer border of the anterior surface of the right side of the epiglottis. On Jan. 7 this was removed by sharp dissection under suspension laryngoscopy. The pathological diagnosis was adenomyxoma with doubtful malignancy. In May, 1941, a biopsy was taken which showed no malignancy."

The examination in 1944 (age 45 yrs.) showed a rather large, yellow, smooth, soft, and lobulated mass on the right side of the tongue extending from the epiglottis to the deep surface of the right tonsil (see Fig. 3). Nothing else of significance was found in the local or general physical examination. On Dec. 7, 1944, under general anesthesia the tumor was removed by sharp dissection along with both tonsils. The tumor had extended between the capsule of the right tonsil and the fossa bed. Bleeding was slight, and the mass whose dimensions were 4.5x3.5x1 cm., was surrounded by a definite capsule and was on the surface of the tongue. The pathological diagnosis by Dr. Warren C. Hunter was submucous lipoma. He commented that this was the first time that he had seen a lipoma in this location.

The wound healed well, and periodic examinations showed no recurrence until June 20, 1952 (age 53 yrs.), when a discrete mass about 1 cm. in diameter, with a broad base, was found on the right side of the tongue in the vallecular fossa. The patient returned for surgery on Feb. 7, 1953. The tumor was now about the size of an olive and was removed by sharp dissection under local anesthesia. A small adjoining mass was also resected.

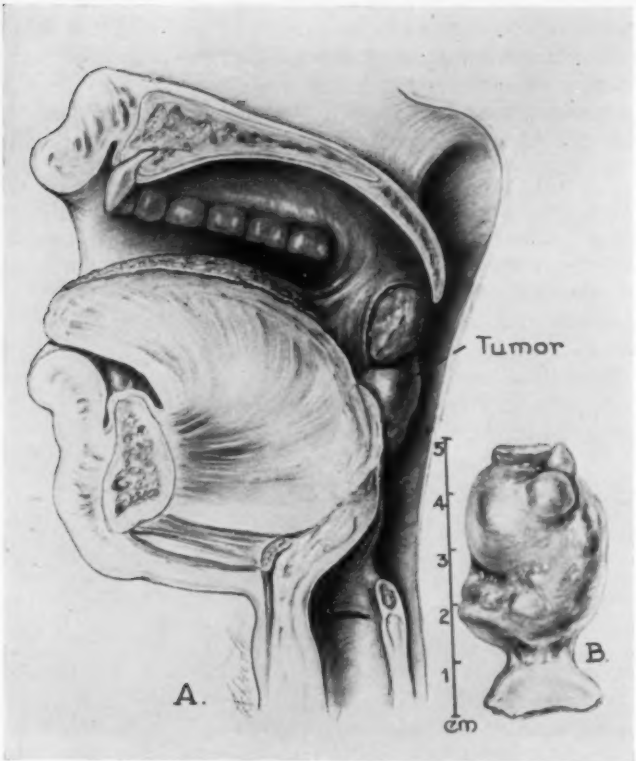

Fig. 3. Case 2. Fibro-liposarcoma of dorsum of tongue extending into tonsil fossa.

The pathology examination, as recorded by Dr. Hunter was as follows: The pathological diagnosis of the tumor removed in 1927 was "adenomyxoma with doubtful malignancy."

Reviewed in late 1957 the histopathology proved to be identical with that observed in the recurrence of 1953, termed recurrent fibro-liposarcoma.

In 1944 a mass of yellow tissue divided into two parts by a cleft surrounded by a thin fibrous zone and measuring 4.5x3.5x1 cm. was reviewed.

Microscopically this proved to be principally fat cells of adult type, together with some mature collagenous type connective tissue. Vascularity was not at all excessive. There were no lipoblastic cells. The pathologic diagnosis was submucous lipoma.

Fig. 4. Case 2. Top—Photomicrograph (X160) of a representative portion of the original tumor removed in 1927. Adult-type fat cells are abundant, but between these in places are elongated cells with fairly large and very deeply stained nuclei, representing the sarcomatous element. Middle—Recurrent tumor of 1944, at this time entirely lipomatous and nonmalignant (X160). Lower—Representative area from the 1953 recurrence, now somewhat more sarcomatous than in 1927, but still containing many adult adipose cells (X160).

In 1953, the specimen consisted of two tissues, one both smooth and rough-surfaced, very soft white to red and measuring 1.3x1x0.5 cm., the other smooth everywhere, soft, white to faintly pink and having dimensions of 3x2.5x1.5 cm. The cut surfaces proved to be white to faintly yellow.

Over a part of the larger tissue was a covering of stratified squamous epithelium along with underlying aggregates of lymphocytes. The mass consisted of connective tissue and of fat, and mingling with the latter were a good many cells of varying size and with hyperchromatic nuclei. The cytoplasm lacked demonstrable vacuolization in the paraffin sections, yet their form otherwise and in a background of adipose tissue gave the impression that this growth must be considered as a recurrent fibroliposarcoma, and such was the pathological diagnosis. Since all of the material had been embedded there was nothing available for fat staining (see Fig. 4).

In a written consultation on this patient, Dr. Simeon T. Cantrell of Seattle, Wish., stated that he had never seen a similar tumor on the tongue, and that it was one of the very rare forms of mesenchymal sarcoma which has considerable radio-sensitivity. He suggested that this patient should not be radiated unless the tumor should recur.

At the present time there is no evidence of local recurrence or metastasis. Obviously this tumor had a low degree of malignancy, as shown by its long duration and its recurrence at one time as a lipoma.

A patient with a simple lipoma on the anterior surface of the tongue has been reported by Braunstein.[6]

Saunders[7] in reporting a lipofibroma of the larynx presents an extensive bibliography on the subject of lipoma of the larynx. Several of the cited cases of primary laryngeal lipoma showed extensions of the tumor to the epiglottis and vallecculae. One tumor originated on the left aryepiglottic fold and was diagnosed as myxolipoma with sarcomatous changes. All of these tumors were removed surgically.

Knowles and Hugill[8] have reported the case of a 12-year-old child who had a primary liposarcoma in the submucosa of the naso-pharynx. The patient died with metastasis to the lungs, liver and brain.

Lipomas are commonly seen in many parts of the body and are usually of no major consequence. Ewing[9] states that "Liposarcomas are most frequent in the intermuscular, peri-

articular, perineal and mediastinal regions. They generally first appear as rounded swellings of spontaneous origin, which grow steadily or rapidly until large or bulky masses result with prominent pressure symptoms. They are usually encapsulated until recurrences develop as diffusely infiltrating, highly malignant and metastasizing processes."

Boyd[10] in discussing retroperitoneal liposarcomas says: "Microscopically the greater part of the mass consists of adipose tissue, but here and there may be found areas of myxomatous tissue and sarcomatous formation. It appears probable that these have developed primarily and that the tumor represents an embryonal type of tissue, rather than that they are due to a transformation of the lipoma."

MIXED TUMOR OF THE NASOPHARYNX.

Case 3. Mrs. D. C. M., white, age 52. In a general physical examination by Dr. Eldon W. Snow on Feb. 10, 1957, a swelling of the left tonsil area was noted. It appeared as a fullness of the soft palate and posterior pillar, which pushed the tonsil forward and medially. The mass did not appear to change in size, and on Oct. 27, 1957, was explored by Dr. Snow. Upon removal of the normal tonsil the tumor immediately protruded through the posterior pillar. It appeared to be of the consistency of cartilage, which fragmented easily and was readily separated from the surrounding tissue. A large fragment was removed for biopsy and the pathological examination by Dr. Warren C. Hunter revealed chondrosarcoma.

X-ray examination by Dr. Arthur L. Hunter revealed a large discrete opacity about 4 cm. in diameter protruding from the left lateral wall of the nasopharynx beyond the midline with no evidence of bone destruction. X-ray examination of the thorax was normal. Otolaryngological examination showed the soft palate and left posterior pillar to be pushed forward by a large mass covered with normal mucosa protruding into the nasopharynx from the left side. It extended almost to the midline and compressed the Eustachian orifice. No other significant abnormality was found.

Definitive surgery was carried out by the writer on Nov. 21, 1957. Under general anesthesia, the left external carotid artery was ligated below the lingual branch. A tracheotomy was performed to facilitate anesthesia, and the hypopharynx was completely packed off. A vertical incision was made through the left side of the soft palate, beginning in the tonsil fossa and extending upwardly to the posterior edge of the hard palate. Tumor tissue with a definite capsule was immediately encountered. It was soon found that the tumor extended into the surrounding muscles extensively. By sharp and blunt dissection, the large neoplasm with a wide margin of surrounding normal muscle was removed in many pieces with a minimum of bleeding. The largest fragment measured 5x4x3 cm. In order to remove the tumor completely it was necessary to resect large portions of the left internal pterygoid muscle, the palatine muscles, and the posterior pillar. The resulting cavity was bounded above by the Eustachian cartilage (not involved by tumor) and

the base of the skull, laterally by the styloid process and carotid sheath, posteriorly by the longus capitis muscle, and anteriorly by the remains of the soft palate.

The post-operative course was uneventful except for trismus which has gradually improved.

The pathological report by Dr. Hunter of the original biopsy was: The portions of tissue first seen had the color, consistency and cutting qualities of hyalin cartilage. Sections contained nothing other than apparent cartilage, and because of the atypical character of the nuclei the initial diagnosis was chondrosarcoma.

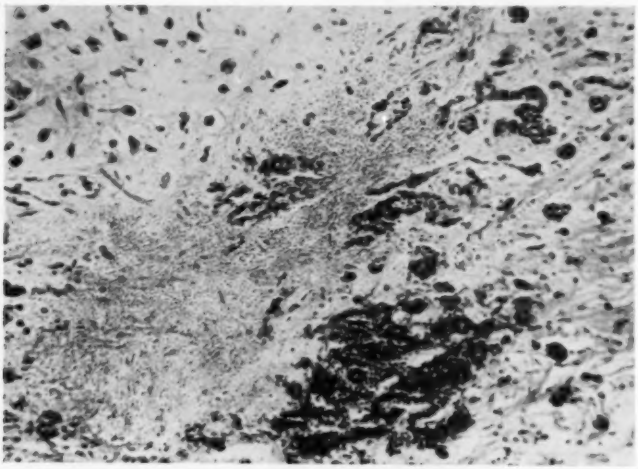

Fig. 5. Case 3. Mixed salivary gland tumor (X160); this field contains many nests of epithelial cells, relatively little matrix and no cartilage, whereas the biopsy consisted entirely of atypical cartilage.

The largest of the masses, removed on Nov. 21, 1957, appeared to be a partially encapsulated lobulated white tissue measuring 5x4x3 cm., cut with the consistency of cartilage and was white to yellow.

Microscopically the growth consisted in large part of bluish-pink to violet staining material, rather poor in cells of apparent epithelial nature occurring in solid strands, and as small alveoli. These had some resemblance to the alveolar epithelium of a serous type of salivary gland. Mucus was not a component of the cells or the lumens of the alveoli. None of the cells appeared to be of squamous type. Embedded in surrounding connective tissue, in close association with skeletal muscle, and entirely away from the tumor were some lobules of a salivary gland of mucous type, unassociated with neoplasm.

In view of the structure of the whole tumor, as contrasted with the fragments making up the earlier biopsy, the original diagnosis of chondrosarcoma was changed to "mixed tumor of aberrant salivary gland" (see Fig. 5).

Boyd[11] states that a mixed tumor has two components, the first of which is the epithelial cell and the second, the spindle or stellate cell, separated by abundant intercellular mucoid material. He states that in places this material may closely resemble or be identical in appearance with cartilage. It was considered very fortunate that this tumor was of the mixed type rather than a chondrosarcoma. Wirth and Shimkin[12] report a chondrosarcoma of the nasopharynx, which was very extensive and had a fatal outcome in spite of extensive surgical removal.

Mixed tumors commonly arise in the major salivary glands, and also occasionally in aberrant salivary glands widely scattered over the whole of the oral mucosa. If the tumor is completely removed by the first operation, the prognosis is generally considered to be quite favorable; however, "recurrence" may follow incomplete removal. In order to be sure of total removal the tissue surrounding the capsule must be resected. Malignant changes are uncommon. The author observed in 1946 the removal by Dr. Louis Clerf of a large mixed tumor of the nasopharynx by a transpalatine approach. In reporting 33 malignant tumors of the nasopharynx, Hara[13] includes two mixed salivary gland tumors.

In the presently reported case, there was no connection between the nasopharyngeal tumor and the deep lobe of the parotid gland; however, Morfit[14] reported 12 mixed tumors arising from the deep lobe of the parotid and presenting in the lateral pharyngeal wall as a large mass covered with normal mucosa. Occasionally also the tumor presented below the horizontal ramus of the mandible. The pharyngeal prominence is to be distinguished from an aneurysm or a large carotid body tumor. External removal of the large parotid mass was recommended.

Sonnenschein[15] has described two mixed tumors in the soft palate. These were surgically removed and are rare in this location.

MALIGNANT LYMPHOMA OF THE TONSIL.

Case 4. Mrs. K. L., white, age 60, consulted her family physician on July 3, 1957, because of a sore throat of two weeks' duration. His exam-

ination revealed the right tonsil to be enlarged and displaced medially. An enlarged anterior cervical lymph gland was present on the same side. Because it was thought to be a peritonsillar abscess, an incision was made above the tonsil, but no pus was found. Under antibiotic therapy, the tonsil decreased somewhat in size. On Aug. 13, 1957, the tonsil was removed and found to be extremely large.

The pathological report by Dr. Jeff Minckler was as follows: The tonsillar tissue is submitted in three pieces, ranging from 2.5 to 3.5 cm. in their greatest dimensions. The epithelial surface is identified on the larger specimen. The surfaces are moderately fragmented, and on sectioning much destruction of the tonsillar tissue is identified. The entire tissue is mottled pale pink to red. Nothing suggestive of a neoplasm is encountered.

Fig. 6. Case 4. At a 40X magnification the contrast between normal tonsil just below the epithelium of the crypt and the malignant lymphoma occupying the lower part of the field is quite evident.

Microscopically the architectural pattern of the tonsil is altered by what is interpreted as a neoplastic process of the lymphoma group. The predominant pattern is that of large follicle-like structures without distinct germinal centers composed of relatively uniform medium sized cells that have somewhat ill-defined cytoplasmic outlines with suggestive tailed processes attached. There is slight to moderate mitotic activity in these fields. In selected areas there is somewhat more dense cellularity with somewhat larger darker staining cells and more apparent mitotic change. There appears to be local infiltration of the capsular surfaces.

Diagnosis: Malignant lymphoma (lymphosarcoma) of the right tonsil (see Fig. 6).

The otolaryngological examination by the writer on Aug. 16, 1957,

showed the soft palate and right pillars to be grossly normal with a gray membrane lining a normal-appearing fossa. In the right side of the neck, anterior to the sterno-cleidomastoid muscle was a freely movable smooth mass of rubbery consistency measuring about 10 cm. in greatest diameter. The remainder of the examination, including an X-ray of the chest, was negative. X-ray therapy was administered in divided doses to the throat and neck. The tumor responded well and examination on Oct. 3, 1957, showed the tonsil fossa to be well healed and the mass in the neck to have completely disappeared.

Malignant lymphomas of the throat and cervical lymph glands are rather common, and are usually considered to be best treated by irradiation.

CARCINOMA OF THE TONSIL.

Case 5. Mr. M. O., white, age 57, sought consultation because of a mass in his neck. He stated that for several months he had visited various physicians in a large clinic because of a sore throat. He was told that he had an infection and was given various antibiotics which gave no relief. Eventually the left side of his neck began to swell and an incision into this mass showed squamous cell carcinoma.

Examination by the writer showed that a fungating tumor about 5 cm. in diameter occupied the left tonsil fossa. The soft palate and pillars were indurated and fixed, and moderate trismus was present. The entire left side of the neck was greatly swollen and indurated. The patient appeared to be in good general health otherwise.

At that time he had already begun a course of cobalt radiotherapy under a competent physician. For that reason, he was advised to continue the treatment, and that if the lesion in the tonsil was controlled, radical surgery of the neck should be considered. His further progress is not known.

Obviously, the prognosis in a patient such as this is discouraging.

Early and small carcinomas of the tonsil can sometimes be completely removed by tonsillectomy. If the tumor is more advanced, irradiation therapy is usually employed. If the metastasis in the cervical lymph glands is not too far advanced, radical neck dissection is usually the best treatment if the primary lesion can be controlled.

SUMMARY.

The records of five patients having tumors in a tonsil or in the surrounding structures are presented. The diagnostic and treatment problems are discussed and the literature is reviewed.

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624 Medical Arts Building.

SINUS SYMPTOMS ORIGINATING FROM MODERN FLIGHT.*

LT. COL. STANLEY H. BEAR,
U.S.A.F. (M.C.)††

With the advent of a new era in aviation, man is ever attaining higher altitudes and traveling at faster speeds. In so doing, he is encountering physical problems which interfere with his best performance and well-being. One of the vital problems is the involvement of the sinuses due to barotrauma, a condition more commonly known as aerosinusitis.

Aerosinusitis is an acute or chronic inflammation of one or more of the nasal accessory sinuses produced by a barometric pressure difference between the air or gas inside the sinuses and that of the surrounding atmosphere. It is commonly characterized by congestion and inflammation of the lining structures. Mucosal or submucosal hemorrhage may occur.[1]

There are two general types of aerosinusitis which may develop: the non-obstructive, which is due to the collection of mucous secretions in the ostei; or the obstructive type, which has three degrees depending upon the severity. Most chronic cases will fall into the latter group and are usually of the third degree.

During the past two years, I have examined, in our clinic, 48 cases of aerosinusitis of the chronic obstructive type. An evaluation has been made of the incidence, predisposing factors producing the physiological disturbances, signs and symptoms, pathology, X-ray findings, treatment and sequelae.

The incidence of aerosinusitis is difficult to evaluate in

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*Read at the meeting of the Middle Section, American Laryngological, Rhinological and Otological Society, Chicago, Ill., Jan. 13, 1958.

†From Surgical Service, 3510th U.S.A.F. Hospital, Maxwell Air Force Base, Alabama.

‡The opinions expressed are those of the author and do not necessarily represent the opinions of any department of the Department of Defense.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 10, 1958.

any group of cases. We were unable to correlate the incidence, because the cases were referred for final disposition after failing to respond to primary treatment. The exact number of acute cases, before becoming chronic, was undetermined. Campbell[2] reported on 35,000 simulated flights in the altitude chamber, where a direct control of all cases was able to be made, and found an incidence of 1.9 per cent. McGibbon[3] noted 18 cases in 1000 chamber flights, or incidence of 1.8 per cent.

One large airline during a one-year period, noted a major discomfort in its passengers from all causes due to air travel, averaged about eight or nine per 1,000.[4] The incidence of sinus and ear distress reported varied from 0.5 to eight per 1,000 passengers carried, and it was felt that this rate would have been higher if all passengers who experienced sinus or ear distress had reported it. Unfortunately, the number of flights made in pressurized aircraft was not differentiated from those in unpressurized aircraft.

To our knowledge, there has been no report made on the incidence of aerosinusitis resulting from jet aircraft flight. Of the 48 individuals we examined, 38 experienced symptoms of aerosinusitis in pressurized jet aircraft. Of the remaining ten cases, six occurred in pressurized conventional aircraft for a total of 44 cases in pressurized aircraft. There were four cases occurring in unpressurized conventional aircraft.

What are the factors producing this change? It was interesting to note that of the 48 aircrew members, there was no direct correlation with the flying time. The minimum number of hours flown by any of the pilots was approximately 50, and the maximum number of hours was over 5,000, when the symptoms became incapacitating. There were four cases occurring in the 0-500 hour group, eight cases in the 500-1,000 hour group, 13 cases in the 1,000-1,500 hour group, 16 cases in the 1,500-3,000 hour group, and seven cases occurring in personnel with more than 3,000 hours of flying time.

The four cases which developed in the 0-500 hour group all occurred during the flight training period in unpressurized conventional aircraft. In all instances the individuals had

been properly briefed on the awareness of difficulty in flight, such as making slower descent and the use of the Valsalva maneuver, especially in cases of Eustachian tube involvement; however, the Valsalva maneuver is of little benefit in sinus obstruction and will not lessen the degree of symptoms. Several fliers with more than 1,000 hours in conventional aircraft experienced symptoms on their very first jet flight. In two instances, symptoms occurred during every descent in jet aircraft; yet they never experienced any difficulty in flying slower conventional aircraft.

There are two main factors which tend to produce difficulty in flight: These are a rapid change in barometric pressure, plus a predisposing factor which interferes with the air or gaseous change between the environment or nasal cavity and that of the sinus cavity. The pressure change at the various altitudes is noted on the following table:[5]

TABLE I.

Altitude Based on United States Standard Atmosphere.
(From Armstrong)

Altitude (Feet)	Pressure (mm. Hg.)
0	760
5,000	632.30
10,000	522.60
20,000	349.10
30,000	225.60
40,000	140.70
50,000	87.30

In general, on ascent from sea level to 5,000 feet, there is a decrease in barometric pressure from 760 mm. Hg. to 632.30 mm. Hg., one-sixth atmosphere, and on ascent to 18,000 feet there is a decrease to 360 mm. Hg., or one-half atmosphere. The change is reversed on descent; therefore, it is especially noted that the greater differrential pressure change is noted at the lower altitudes. This effect is more pronounced as the speed or rate of change is increased and greatly altered if a predisposing factor in any way interferes with this change. Proetz[6] states that a reduction of one-fifth atmosphere (152 mm. Hg.) pressure is ordinarily well tolerated by the mucosa,

but when in much excess of this may be expected to be followed by hemorrhage, either mucosal or submucosal. Behnke[7] found that if a sinus opening is obstructed and an increased pressure of one to two pounds transmitted to the body tissues and circulating blood, an area of relatively decreased pressure is produced in the air spaces. This results in a cupping action in the cavity and a vascular congestion of the mucous membrane. Tension is produced on the cells of the living membrane, and the blood vessels distributed in tissue surrounding the occluded space. Pain is present in and around the involved area, associated with congestion and hemorrhage.

Today most modern pressurized aircraft fly at a cabin altitude of approximately 8,000, and those of future jet operations will be at or near this altitude on long flights. There is only a slight difference on ascent or descent of selected cabin altitude from that of the plane; therefore, from 8,000 feet to ground level, pressurization will have little beneficial effect on the individual crew member or passenger. At the present time C.A.A. Regulations allow a maximum rate of change of cabin altitude equivalent to approximately 300 feet per minute.[8] This applies to both ascent and descent. In future jet aircraft operations there are times when a faster rate of descent may be produced through loss of pressurization, faulty pressurization valves, instrument approaches, or in cases of fuel starvation. Any one of these factors will necessitate a more rapid rate of barometric pressure change, and added to a predisposing intrinsic factor in the passenger or crew member, sinus symptoms will likely result.

It is worthy to note at this point that there is a difference in the pressurization between military jet aircraft and those of anticipated commercial design. At lower levels, the altitude of pressurization is approximately 12,000 to 13,000, rather than 8,000 feet, and the differential pressure at high altitudes is usually the maximum of 5-plus pounds per square inch, while the commercial planes are designed for 8-plus pounds per square inch for operation.

In all the cases in our series, there were only a few occurring above 15,000 feet cabin altitude. The majority occurred

between 12,000 feet and ground level. Some occurred at an altitude of less than 5,000 feet. The condition seemed to be aggravated by the rate and duration of the pressure change.

There are many predisposing factors which, in combination with a rapid barometric pressure change, either on ascent or descent, will produce an acute obstruction to the sinus passage with resulting aerosinusitis. The more common, in decreasing order of occurence, are:

TABLE II.

Predisposing Factors in Etiology of Aerosinusitis.

Deflection of Nasal Septum (More than 50%)	23
Hypertrophy of middle turbinate with encroachment of middle meatus and nasofrontal duct orifice	14
Nasal allergy	10
Upper respiratory infection	7
Polyps in middle meatus	7
Mucocele	2
Septal spur	1
Hypertrophic rhinitis	1
No abnormal finding at examination	1
Cyst on floor of maxillary sinus	1
Vasomotor rhinitis	1
Osteoma over sinus orifice of nasofrontal duct	1

In some cases it was noted that more than one factor was prevalent, such as allergy and nasal polyps, or deflection of the nasal septum with encroachment of the middle turbinate on the nasofrontal duct; thus, the total on the above chart will, in some instances, have more than one etiologic factor. It is important to note that deflection of the septum was high in over two-thirds of the cases. This resulted in the impingement of the middle turbinates laterally over the opening of the nasofrontal duct with encroachment on the duct orifice. This was a significant finding in nearly 50 per cent of the cases. The one case of osteoma was located at the orifice of the superior part of the nasofrontal duct and caused almost complete collapse of the mucosa lining the duct. This acted as a suction valve on descent and would obstruct the orifice, producing severe symptoms. Nasal allergy and polyps were present in seven of the cases. They were obstructing the middle meatus in all instances. The case of vasomotor rhinitis

was in a cadet in training period, and was aggravated by an emotional problem. On flying, his nose would completely obstruct, causing pain in sinus. He was recommended for psychiatric evaluation and elimination from flying duties.

The above conditions were present even in a select group of individuals. Aircrew members were given rigid acceptance physical examinations; however, in most instances the deformities were acquired or developed during their career. In addition, most pilots had been indoctrinated in the preventive medical aspects of flying and proper care of themselves during flight. The average air passenger is not as well indoctrinated in active and preventive measures that can be performed while flying. Many prospective passengers may have a nasal allergy of sufficient degree to be symptomatic on descent; active sinus pathology; upper respiratory infection; nasal obstruction of one sort or another, or a marked degree of apprehension and anxiety. All of these act as predisposing factors, and when subjected to a rapid change of descent will produce aerosinusitis of one degree or another. On ascent, the predisposing condition is basically intra-sinus, or in the osteum or duct of the sinus, while on descent the difficulty present is in the nasal cavity or surrounding structures in the area of the duct orifices leading into the sinus, as noted in the previously mentioned causative factors. From these findings one is able to deduce whether the difficulty will be encountered on ascent or descent, or both.

There are many symptoms produced by aerosinusitis, but the predominant symptom in most cases is pain located over the involved sinus. It is described as being sharp, lancinating in character, non-radiating and occurring most often during descent. The frontal sinus was involved in 23 instances, the maxillary sinus in seven, and both frontal and maxillary in two instances (see Table III).

The second most common symptom was that of nasal obstruction. This occurred in nine cases and was almost equally divided, with involvement of either the frontal or maxillary sinus. There were seven cases of pain on both ascent and descent, with three associated with involvement of the frontal

sinus and four with both frontal and maxillary sinuses involved. The fourth major symptom was that of a feeling of pressure over the involved sinus, and occurred in seven cases. The fifth predominant symptom or sign was evidence of a delayed bleeding, usually of old blood, occurring 24 or more hours after primary involvement. This was noted on expectoration upon arising in the morning, or on blowing the nose, when blood-streaked mucous would be present. The lesser symptoms are noted on the Table above.

This discussion does not include the ethmoids and sphenoid sinuses, because no case of aerosinusitis occurred where only

TABLE III.

Subjective Symptoms Experienced with Reference to Involved Sinus.

	Frontal	Maxillary	Frontal and Maxillary
Sharp pain from descent	23	7	2
Sharp pain from ascent	0	1	0
Pain from ascent and descent	3	0	4
Generalized headache	2	0	3
Pressure over sinus	3	2	2
Nasal obstruction	4	3	2
Bleeding (delayed)	4	0	2
Blurring of vision	3	0	0
Lacrimation	2	0	0
Swelling of upper eyelid	1	0	0
Rhinorrhea	0	1	1
"Plugged" ears	3	0	1

the ethmoid and sphenoid was primarily involved. When there is severe involvement of either maxillary or frontal sinuses there may be associated involvement of the ethmoid sinus, but it is not felt to be the cause of the primary symptom complex. This was also the finding of other authors.[2,3] As previously noted, the frontal sinus was most frequently involved; next, the frontal and maxillary, with the maxillary sinus the least involved. Campbell and McGibbon[2,3] also found the frontal sinus to be the most frequently involved, while Wright, in his series, found the most frequent site of involvement to be the maxillary.[9] The reasoning for greater involvement of the frontal sinus is based on the greater length of the nasofrontal duct and its tortuous path and small caliber

of opening. The opening of the maxillary sinus is larger and may have accessory openings. Von Dirringshofer[10] states that more involvement is expected in individuals with large frontal sinuses, but in one of our cases with severe frontal pain there was only a small frontal cell and no development of the frontal sinuses.

X-ray examination is an important phase in the evaluation of aerosinusitis. When possible they should be taken immediately as well as several days later if symptoms and signs persist. It has been noted in cases where there is either mucosal or submucosal bleeding that definite delineation of the area involved may not be demonstrated until the bleeding is organized into a hematoma. One of the most difficult X-ray diagnoses to make is submucosal hemorrhage. It is very helpful to have previous X-ray films to help differentiate from polypoid hypertrophy or polyps. The latter is uncommon in the frontal sinus; otherwise, the clinical history must be evaluated to rule out the presence of nasal polyps and allergy. This was the opinion of Coche[11]; also in four of our cases of bleeding with hematoma found on X-ray. The views used in routine evaluation are Caldwell, Waters, and lateral views. When indicated, laminograms are beneficial in making a differential diagnosis (see Table IV).

There were two cases in which more than one finding was made. The pathology of aerosinusitis varies, depending on the etiologic factor and the degree of intra-sinus or naso-frontal duct changes. The histologic structure of the mucosa is a continuation of the nasal respiratory mucosa with slight variations. The submucosa is thinner and tends to blend with the fibrous periosteum of the underlying bony sinus wall. The epithelium is a thinner cuboidal-like, stratified ciliated structure. The resulting sinus mucosa changes can vary from thickening of the mucosa, either inflammatory or polypoid, or there can be presence of fluid, blood, mucous, or purulent secretions in the chamber of the sinus. In some cases, mucoceles will be present while others have osteoma in the cavity covered with a very thin mucosa. Probably the most marked change is that noted in rapid changes of barometric pressure with the complete occlusion of the sinus

orifice. This results in free bleeding from the mucosa surface or submucosally, producing submucosal hematoma. This was noted in four of our cases. Campbell was able to demonstrate the same findings in his experiment with a dog in an altitude chamber.

The treatment of aerosinusitis usually falls into one of two categories: the acute phase, and the chronic or persistent

TABLE IV.

X-ray Findings in Aerosinusitis.

FRONTAL.	
Clouding frontal	19
Submucosal hemorrhage	5
Polypoid hyperplasia of sinus mucosa	3
Mucocele with hemorrhage	2
Osteoma at orifice of nasofrontal duct	1
Large ethmoid cell at orifice of nasofrontal duct	1
No sinuses present	1
Fluid level	1
No abnormal findings	1
MAXILLARY.	
Clouding	5
Polypoid hyperplasia	3
Retention cyst floor of maxillary sinus	2
BOTH FRONTAL AND MAXILLARY.	
Clouding	8
Hyperplastic scarring	2
Polypoid mucous membrane	2

type. The acute stage is treated conservatively with treatment initiated as soon as possible after symptoms occur. Treatment consists in the use of vasocontrictor sprays and packs, steam inhalation, antibiotics prophylactically, and anodynes for control of discomfort. In severe cases, applicators of 5 per cent cocaine and 1 per cent ephedrine are placed directly in the middle meatus at the opening of the nasofrontal duct of the side involved. Depending on the time interval following initiation of the first symptoms, most cases will respond to conservative management. It is worthy of mention that the longer the interval before treatment is begun, the more difficult it is to obtain patency of the involved sinus.

In our series, almost all the cases were of a chronic nature, having been referred when conservative management failed, or when there was a recurrence of the condition at frequent intervals. Following is a table showing the various treatments utilized in the 48 cases of our series.

In several cases, more than one procedure or combined procedures were necessary to correct the condition. In 20 cases, conservative management was used which consisted of antibiotics when indicated, vasoconstrictors and steam inhalation, or minor surgical manipulation to produce a patent osteum of the involved sinus. The conservative treatment

TABLE V.

Treatment of Aerosinusitis (Chronic Cases).

Conservative (antibiotics, vasoconstrictors, steam inhalation)	20
Submucous resection	19
Infraction of middle turbinate	13
Polypectomy	7
Allergic management	5
Frontal sinusotomy (Lynch approach)	4
Antral window	1
Vasomotor rhinitis*	1

*Vasomotor rhinitis—due to emotional disturbance. Recommended elimination from flying duties.

was mainly combined with infraction of the middle turbinate which was necessary in 13 cases, and in cases of post-polypectomy to maintain normal drainage; however, there were 19 cases in which it was necessary to perform a submucous resection (Killian type). In all but seven of these cases, the obstruction was located in the region of the middle turbinate, and in several cases following resection, infraction of the middle turbinate was combined in the treatment. Four cases of frontal sinusotomy, by the Lynch approach, were necessary to remove the frontal sinus pathology and obtain a patent nasofrontal duct. Allergic management was utilized in five cases with fair results. In one case of an antral cyst, an antral window was performed with curettement of the cyst. Two cases of recurrent purulent infection of the sinus were treated by irrigation; one was of the frontal sinus; the other, of the maxillary sinus. Two cases were surgically treated by

partial turbinectomy of the anterior third of the middle turbinate because of polypoid hypertrophy encroaching on the opening of the nasofrontal duct. There was one case of vasomotor rhinitis which was due to an emotional disturbance, and it was recommended that the individual have psychiatric evaluation and be eliminated from flying duties.

As a group, chronic cases of aerosinusitis must have treatment directed to the precipitating factor, and in most instances the condition can be corrected only by surgical or allergic management. It is easy to ascertain that conservative management will be of little avail where these conditions exist. Many of the predisposing factors would not require surgical intervention if the individual were not flying; however, when required to fly either as passenger or aircrew member, surgical correction is necessary to alleviate the condition.

Of the 48 cases, there were three cases which were entirely unsuccessful following treatment: two of these cases showed marked nasal polyposis and polypoid hypertrophy of the turbinates and an underlying allergic condition. The individuals were unable to fly without repeated bouts of aerosinusitis. The other case was that of the vasomotor rhinitis, for which no treatment was indicated as it was felt to be due to an emotional basis. There were three cases in which the individuals were able to fly, but at intermittent intervals would still experience symptoms, usually not to the degree of incapacitation from flying duties. Two of these cases were allergic rhinitis with polyposis, while the third case was that of recurrent upper respiratory infection. This individual is still undergoing treatment to determine the etiology of his infection. The remaining 41 cases have been returned to flying duty and have experienced no difficulty in most cases, and minimal symptoms in a few cases which did not interfere with duty performance. All cases were followed for a minimum of one year; however, due to loss of contact and follow-up in some cases the incidence of recurrence may have been slightly higher. One case has recently been discharged from the hospital following frontal sinus surgery for submucosal hemorrhage and obstruction to the nasofrontal duct. He has

not been returned to flying duty, but exhibits a patent duct at the present time and will be given trial indoctrination in the altitude pressure chamber to see whether his results have been successful before returning to full flying duties.

In all cases of aerosinusitis, treatment is directed to removing the etiologic factor as well as the existing condition.

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MANAGEMENT OF SINUSITIS IN CHILDREN.*

CLIFFORD F. LAKE, M.D.,†

Rochester, Minn.

Sinusitis, either alone or associated with some other inflammatory process in the respiratory tract or related areas, occurs frequently in childhood. Although this fact has been demonstrated and reported many times in the past, it seems that in this age of antibiotics its re-emphasis is indicated.

Dependence on antibiotics alone, and especially on short courses of them, during acute respiratory tract infections may only modify or mask associated acute sinusitis.

Failure to examine the nose and nasopharynx completely when possible, or to obtain roentgenologic views of the paranasal sinuses, often causes failure in diagnosis. Birdsall[1] stated that nasal sinus infection in children is common, but is often overlooked because of its usually insidious onset, the lack of definite symptoms, and the difficulty of examining the affected parts.

From the ages of four or five most children who are cooperative can be given satisfactory nasal examination. Children this old frequently will be co-operative on nasopharyngeal examinations with the small to medium-sized mirrors. A brief explanation of what is about to happen, and a demonstration of quiet rhythmical breathing often will convert a skeptical child into a most co-operative youngster. If the tongue depressor and nasopharyngeal mirror are introduced simultaneously, the number of satisfactory nasopharyngeal examinations will be increased. This method of introducing the instruments relieves the child of the "double jeopardy" of separate instrumentations.

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*Read at the meeting of the Middle Section, American Laryngological, Rhinological and Otological Society, Chicago, Ill., Jan. 13, 1958.

†From the Section of Otolaryngology and Rhinology, Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 10, 1958.

Obviously the anatomic development of the paranasal sinuses in accord with age has an effect on the sites in which infection may develop. In older children the roentgenologic views are particularly helpful in localizing the site of infection. Maresh,[2] in a study of routine roentgenograms of the sinuses of 100 children who were followed from birth to maturity, observed many variations in the size and shape of antrums, ethmoid cells, and frontal sinuses.

DIAGNOSIS AND SYMPTOMATOLOGY.

Sinusitis should be included in the differential diagnosis when symptoms of prolonged rhinorrhea, coughing, frequent sniffling, or throat clearing are present. A history of recurrent respiratory infections, otitis media, secretory otitis, and sore throats should suggest the possibility of the presence of sinusitis. Wishart[3] stated that the most frequent complaint in an otherwise healthy child in whom sinusitis is discovered is repeated head colds.

In cases of small children the history obtained from the parents and the objective findings on examination, with the findings from roentgenologic examination in some instances, usually will suffice in making the diagnoses. Older children often can contribute to their history, and should be encouraged to do so. Frequently such children describe their symptoms accurately, especially when pain is present in acute sinusitis or in a flare-up of chronic disease.

In the history, allergy affecting either the patient or his family should not be overlooked.

MANAGEMENT OF ACUTE SINUSITIS.

The best basis for management of acute sinusitis in children is consideration of the child as a whole and the severity of his infection. A culture of the nasal secretions should be made and tests of sensitivity to antibiotics performed. The child's reaction to obtaining the material for culture from his nose may indicate the degree of co-operativeness to be expected in carrying out local treatment. If the infection is severe enough to warrant the use of antibiotics one may wait

several days before attempting therapy directed to the nose. Antihistaminic drugs are of particular value in cases with a history of vasomotor or allergic rhinitis.

Treatment directed to the nose usually is a matter of personal choice. Prolonged use of vasoconstrictors often produces the familiar rebound phenomena. Application of argyrol packs to the middle meatus and in the nasal chamber, followed by gentle suction, may prove of value in the removal of purulent secretion from the nose and sinuses. In some children lavage of the maxillary sinus via the natural ostium can be carried out after the acute edematous stage has subsided.

In an instance of acute ethmoiditis with external manifestations, as is found occasionally in a young child, surgical drainage may be necessary; however, if seen in the early stages this situation will usually respond to antibiotic treatment.

Heat applied to the area over the involved sinus or sinuses may afford some relief of pain; however, sometimes it may exacerbate the pain. Increasing the humidity in the patient's bedroom and making him comfortable are beneficial in his general care.

During his course of treatment the patient should be reexamined for conditions which may have influenced the onset of the sinusitis or the application of therapeutic measures. Enlarged adenoids, septal deflections, and allergic changes in the nasal mucosa are commonly occurring factors predisposing to acute sinusitis.

The status of the patient's general health should be investigated. Whatever general measures are necessary to improve it should be carried out; also it may be necessary to remove enlarged adenoids or a septal deflection to gain final resolution of the disease.

Roentgen therapy has been used successfully in the treatment of acute or subacute sinusitis in the past. Laing[4] reported treatment with Roentgen therapy in 900 cases of subacute sinusitis in children, 639 of whom were cured during

one series of treatments; however, with the knowledge of risks involved in exposure to radiation I prefer not to use it for children.

MANAGEMENT OF CHRONIC SINUSITIS IN CHILDREN.

Adequate treatment may include several types of therapy, and the physician should not limit himself to an inflexible routine; rather, he should avail himself of all procedures, and use them as the situation demands, to restore the paranasal sinuses to their normal physiologic state.

The treatment of chronic sinusitis follows the same principles in using antibiotics and intranasal therapy which were mentioned in the treatment of acute sinusitis. Lavage of the maxillary sinus via its natural ostium can be accomplished in many older children.

If surgical treatment is necessary it usually can be done intranasally, except for most instances in which external manifestations exist.

The patient's general health should be evaluated. A search for predisposing factors should be made, and their eradication accomplished, and again, the history of allergy in the patient or his family should not be overlooked. The rather special problem of chronic sinusitis, associated with bronchiectasis, is not within the scope of this presentation, but the possibility of a co-existing bronchiectasis must be ruled out in all cases of chronic sinusitis.

REPORT OF CASES.

Case 1. An 11-year-old boy was registered at the Mayo Clinic on Sept. 26, 1957. His complaint was that sneezing, itching of the eyes, and nasal discharge had persisted for one month. After a general examination in the Section of Pediatrics, an ear, nose, and throat examination was requested.

In the Section of Otolaryngology and Rhinology, examination of the ears revealed no abnormalities. Examination of the nose showed much thick, sticky mucoid discharge on both sides, particularly on the left, some of which was yellowish and obviously mucopurulent. The turbinates were moderately congested on both sides. Pus was demonstrated coming from the natural ostium of the left antrum. The tonsils had been removed. There was a thick mucoid discharge in the nasopharynx, along with some mucopurulent material.

Roentgenograms of the sinuses showed thickening about the maxillary

sinuses and ethmoids, particularly the left antrum. Skin tests were made which showed sensitivity to cat hair, cattle hair, goat hair, house dust, mixed feathers, pyrethrum, and giant and small ragweed.

A diagnosis of allergic rhinitis and sinusitis was made. The treatment consisted of giving the child an antihistaminic drug for two weeks, after which he was re-examined. The nose then appeared to be normal, with no evidence of any unusual discharge. New roentgenograms showed marked clearing of the paranasal sinuses.

Comment. This case is an example of sinusitis superimposed on hay fever, and illustrates the improvement which occurred from the use of an antihistaminic prescription during the recession of the hay fever season. Further care in regard to the allergic manifestations was advised.

Case 2. A seven-year-old girl was registered at the clinic Nov. 8, 1957. She had had pneumonia approximately two months prior to her registration. After five days in the hospital and two weeks at home for recovery, she had returned to school; but after two days in school, left otitis media developed. To combat this ear infection, she had been given antibiotics for a few days. (The exact type of antibiotic and the number of days that it was given could not be determined at the clinic.) Approximately a week later the patient developed a sore throat, and a course of penicillin had been administered.

On Oct. 30, the child had been seen in consultation by an oto-rhino-laryngologist. A diagnosis of sinusitis had been made, nasal irrigations had been carried out, and she had seemed to improve. On Nov. 6, however, she complained of a sore throat again, and her trip to the clinic followed.

She was first examined in the Section of Pediatrics. Only normal findings resulted from general examination, a roentgenogram of the lungs, blood counts, urinalysis, and electrophoresis of serum protein.

The child was examined in the Section of Otolaryngology and Rhinology, and her eardrums were found to be intact, although the left showed slight infection from the recent acute otitis media. Her hearing of tuning forks seemed normal. The nose looked clear on examination, and no pus was visible on anterior rhinoscopy. The turbinates were only slightly congested. Roentgenograms of the child's sinuses showed involvement, particularly of the antra, and especially on the left. The tonsils had been removed and the nasopharynx contained only a minimal amount of lymphoid tissue. A culture of the nasal secretion was negative.

It was thought that this patient's disorder was sinusitis, the diagnosis depending largely on the history and on the appearance of the roentgenograms of the sinuses. A two-week trial of antibiotics was prescribed, and the patient was advised to return at its conclusion.

She did return at that time. On examination her nose appeared normal, and in roentgenograms her sinuses seemed clear. The child was feeling well.

Comment. This is a case of recurring upper respiratory infection, sore throats, and otitis media, in which a diagnosis of sinusitis was made largely on the history and roentgenographic findings. The response to a two-week course of an

antibiotic was excellent, and the roentgenograms which were taken at the end of this treatment illustrated marked clearing within the sinuses. There was no history of allergy in the family of the patient.

Case 3. A six-year-old boy was registered at the clinic Nov. 28, 1956. He had had persistent nasal obstruction for at least a year or so. Infections of the upper part of the respiratory tract had been frequent, and he had had pneumonia in 1952. There was no history of a definite allergic background, but the patient's mother had had mild vasomotor rhinitis.

In the general examination nothing out of the ordinary was found except mild obesity. He was referred to the Section of Otolaryngology and Rhinology. Our examination revealed the tympanic membranes to be intact, but they appeared slightly dull, and his hearing seemed to be reduced very slightly through the entire range in both ears. An audiogram showed very slight bilateral conductive deafness. On nasal examination the nasal airways seemed to be open; however, mucus was pooled on the nasal floor, and when this was removed the adenoid could be seen obstructing the choana. The tonsils were enlarged (grade III, on a basis of 1 to 4, with one being slightest), and combined tonsillectomy and adenoidectomy was advised. The operation was carried out Nov. 30, 1956, and large masses of adenoid, and the tonsils were removed.

The patient then got along very well until three weeks later, when he developed an upper respiratory infection. On examination a large amount of pus was observed in each nasal passage. A culture of the discharge was reported as showing usual flora. Tetracycline (achromycin) was prescribed, and the child's nose cleared nicely after a ten days' trial.

The patient was again seen on January 23, 1957, with a report that a cough had developed two days previously, and he had coughed most of the night prior to his examination. His case was followed for several weeks; but the situation did not clear, and a culture again was made of the nasal secretions, this time showing many Hemophilus influenzae organisms. Electrophoresis of serum protein gave findings within normal limits. Roentgenograms of the sinuses showed cloudiness throughout all of them. The patient was given a preparation of streptomycin with dihydrostreptomycin (distrycin) 500 mg. twice daily for ten days, and therapy directed to the nose was begun with shrinkage and suction and application of argyrol packs to the middle meatus on each side. This was carried out for two weeks, after which the nose was clean, the child was breathing freely, and roentgenograms of the sinuses showed marked improvement, there being only a minimal amount of thickening in the left antrum. Clinically, the child was well.

The case has been followed since; and during one more upper respiratory infection the boy has gotten along well, with no further recurrences of his sinusitis.

Comment. This case illustrates the need in many instances of treatment to the nose as well as antibiotics in order to clear a sinusitis.

Case 4. A 4½-year-old girl was registered at the clinic May 3, 1956. A history of repeated prolonged colds, worse in the Winter months, was obtained. She had less difficulty during the Summer, and her symptoms were not related to the hay fever season. There was a history of allergic

rhinitis on the paternal side of the family. The child had been taking an antibiotic of an undiscoverable type for 11 days prior to her admission for examination.

On examination of the ears, the tympanic membranes were intact with very slight redness on the right. The nasal septum was straight. There was mucopus on both sides of the nose. The tonsils were of size 2 on the basis of 1 to 4. The nasopharynx was examined with a mirror, and a good view was obtained; it was noted that an adenoid blocked most of the choana. On roentgenograms of the sinuses both antra were diffusely cloudy. A culture of the nasal secretions was reported as showing usual flora.

The patient was given penicillin for ten days. At the end of that time the nose looked clear, and no discharge was noted on examination of the nasopharynx. Roentgenograms showed clearing of the paranasal sinuses.

Arrangements were made for removal of the tonsils and adenoids; this was done June 15, 1956. After the operation the child got along well and was dismissed June 21, 1956.

The child was observed a year later during the course of a common cold; however, the cold cleared in eight days without complications or recurrence of sinusitis.

Comment. This case is an instance in which an enlarged adenoid obstructing the choana seemed to play an important part in the persistence of the patient's nasal symptoms and sinusitis.

SUMMARY AND CONCLUSIONS.

The occurrence of sinusitis in childhood deserves re-emphasis. A short course of antibiotics may only modify or mask an acute sinusitis during the course of an upper respiratory infection. Examination of the nose and nasopharynx should be complete when at all possible. The value of roentgenograms and the history obtained from the parents and the child is great.

The foregoing diagnostic principles and a discussion of the management of acute and chronic sinusitis in children have been illustrated in reports of four cases.

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ANATOMICAL OBSERVATIONS FOR RHINOPLASTIC SURGERY.*

ROMEO A. LUONGO, M.D.,
and
ROMEO A. LUONGO, JR., M.D.,
Philadelphia, Pa.

ANATOMIC RELATIONSHIPS AT THE NASION.

The squama of the frontal bone projects downwards, at the midline, to form the *nasal part*. The lower margin of the nasal part, which is beveled, rough, uneven, and serrated, is called the *nasal notch*. The *upper margins* of the nasal bones and of the frontal processes of the maxillae imbricate, like shingles, upon the beveled portion of the nasal notch.

From the center of the nasal notch, the *nasal process* projects downwards and forwards underneath the upper margins of the nasal bones and the frontal process of the maxillae. The *frontal spine* extends below farther than the nasal process itself, and it becomes fused with the perpendicular plate of the ethmoid posteriorly, and with the septal crests of the nasal bones anteriorly to give the bridge of the nose a solid support.

This type of construction makes infracturing difficult and inadvisable. Out-fracturing is the procedure to be chosen.

If, to complete an osleotomy, a chisel is used to fracture at the nasion, the surgeon should bear in mind that this technique may cause fractures of the sloping edges of the nasal notch, of the frontal spine, of the perpendicular plate of the ethmoid, destroying the main support of the nasal bridge, and the impact of the chisel may even be transmitted to the cribriform plate; furthermore, when the frontal sinus extends below the fronto-nasal suture line, which is located at the

*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., Philadelphia, Pa., Jan. 9, 1958.
Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 10, 1958.

nasion, the chisel may force the ends of the fractured bones into the lumen of the sinus, causing frontal sinusitis.

FRONTAL SINUS RELATIONSHIPS.

At the *nasal notch* of the frontal bone, the floor of the frontal sinus is normally at the level of the glabella, but in some cases, it may extend as far down as the middle-third of the nasal bones (over-developed sinus), or it may stop short of the glabella (under-developed sinus).

OVERDEVELOPED FRONTAL SINUS.

When the frontal sinus extends beyond the glabella, its floor is attached directly to the inner surfaces of the nasal bones, which in this instance become part of the anterior wall of the sinus (see Fig. 1).

Thus, manipulations of the nasal bones at the nasion may injure the frontal sinus. A natural protection is given by the fact that the periosteum, which covers the posterior surface of the anterior wall of the frontal sinus, is tough and loosely attached. Its toughness gives it a good deal of resistance to cutting by sharp instruments, and its looseness renders it easily detachable at the slightest pressure. These two favorable conditions form a very valuable protection against accidental entry into the lumen of the sinus when an ostectomy is performed.

X-ray studies will give valuable information about the size and extent of the frontal sinus and its relationship to the nasal bones.

UNDERDEVELOPED FRONTAL SINUS.

When the frontal sinus is not well-developed, it will stop short of the glabella, and a block of compact bone will be found where the sinus is normally located. At the nasion this block of compact bone fuses with the nasal bones, the frontal processes of the maxillae, the bony nasal septum, the frontal spine and the nasal notch of the frontal bone, to form a huge block of solid bone, which will make the ostectomy a difficult procedure.

Fig. 1. Over-developed frontal sinus. The floor of the frontal sinus is about 3 cm. below the upper ends of the nasal bones which become part of the anterior wall of the sinus.

Before rhinoplastic surgery is considered, X-ray studies of this region should be made. These should include the frontal sinus, the nasal bones and the frontal processes of the maxillae. The naso-frontal suture line should be mapped out, and its relationship to the frontal sinus and to the above-described block of bone should be determined. This suture line originates at the nasion, where it runs straight across the upper limits of the nasal dorsum and, when it reaches the lateral

Fig. 2. Under-developed frontal sinus. The floor of the frontal sinus is about 2 cm. above the upper ends of the nasal bones. A solid block of bone is found where the sinus is normally located.

nasal walls, it turns downward, outward and backward, pointing directly to the inner canthi of the eyes (see Fig. 2).

THE PRE-LACRIMAL GROOVE.

One of the most important landmarks of the nasal pyramid is the inner rim of the orbit. This is essentially the anterior lacrimal crest. Adjacent to it, medially, is a groove which is semi-lunar in shape and runs parallel to the inner orbital rim. We shall call this groove the pre-lacrimal groove (sul-

cus pre-lacrimalis). This landmark is important, because it indicates the posterior limits of rhinoplastic surgery.

Posterior to this groove are found the following vital structures: the lacrimal sac, the naso-lacrimal duct, the orbital contents, the agger nasi cells, the middle turbinate, the cribriform plate and the naso-frontal duct.

SUMMARY.

1. The frontal sinus may be over-developed and extend downwards beyond the glabella, past the fronto-nasal suture line, reaching as far down as the middle of the nasal bones. The nasal bones themselves will then become part of the anterior wall of the frontal sinus, and the floor of the sinus will be attached directly to the inner surfaces of the nasal bones.

2. The frontal sinus may be under-developed and stop short of the glabella. In this instance, a solid block of compact bone will be found where the sinus is normally located. X-ray studies will determine the relationship of the frontal sinus to the nasal bones.

3. The nasal notch of the frontal bone is beveled, at the expense of the outer layer, to form a suitable bed for the imbrication of the upper edges of the nasal bones and of the frontal processes of the maxillae. The frontal spine extends further downward from the center of the nasal notch, to become fused with the septal crests of the nasal bones and with the bony septum, to give the nasal bridge greater support. This construction makes infracturing impracticable.

4. The pre-lacrimal groove (sulcus pre-lacrimal) is one of the most important landmarks of the nasal pyramid. It is located between the inner orbital rim and the postero-lateral margin of the nasal pyramid. It runs parallel to the inner orbital rim, and it is semi-lunar in shape. It can easily be felt by digitation. This groove indicates the posterior limits for rhinoplastic surgery. There are no vital structures anterior to this groove; they are all posterior to it.

2054 Locust Street.

THE USE OF ANTIBIOTIC-STERIOD COMBINATIONS AS NEBULIZING AGENTS IN THE TREATMENT OF LARYNGEAL CONDITIONS.

A Preliminary Report.*

BERNARD L. SILVERBLATT, M.D.,

Pittsburgh, Pa.

The treatment of disease entities affecting the larynx is predominantly surgical. Except for antibiotics in the acute infectious reactions and streptomycin in tuberculous laryngitis, other therapeutic measures have failed to produce the enthusiastic results portrayed by their author. Thus podophyllin, hormonal agents as theelin, adrenalin, etc., are seldom prescribed today. Limitations of vocal abuse, smoking and alcohol, persist as standard directives to the patient with hoarseness.

For almost two years, we have been using antibiotic steriod combinations in the treatment of many of our laryngeal problems. This combination has been used in nebulization form, both as a primary therapeutic agent and as an adjunct to surgical procedures. We feel that the combination has rational and merit for usage. I would stress, however, that they are not "cure-alls."

ANALYSIS OF THE PROBLEM AND RATIONAL OF THERAPY.

Standard textbooks list[1] better than 50 specific diseases of the larynx. Standard nomenclature increases this to better than 150 diagnoses. This discrepancy is partially due to our own inadequacies in standardizing nomenclature for disease entities; thus a clinical entity may be diagnosed on the basis of the etiology, the pathologic picture, the anatomic deformity or disability, and with the name of an author.

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*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., Philadelphia, Pa., Jan. 9, 1958.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Feb. 27, 1958.

If we analyze the laryngeal diseases, however, we find that they can be classified into six categories: *1.* Congenital Defects; *2.* Infections; *3.* Toxic; *4.* Traumatic; *5.* Paralysis, neuromuscular and systemic disturbances; *6.* Tumors.

Of these, congenital defects and paralysis can be eliminated from this discussion. The others may be considered, since they have a common factor in that they represent an inflammatory reaction, using the term inflammatory as a nonspecific reaction to injury whether due to infection, allergy, irritation or trauma. The inclusion of tumors depends upon these factors: *1.* Many of the so-called benign tumors of the larynx are actually non-neoplastic in origin; *2.* Malignant tumors are of unknown etiology and steriods, and antibiotics have been used to control neoplastic activity in other parts of the body; *3.* Nebulization might be a useful adjunct in the postoperative healing program.

The pathologic response to inflammation in the larynx is the same as elsewhere in the body, differing only in the symptomatology of the local anatomy. The inflammatory reaction takes place in the subcutaneous connective tissue.[2,3] This reaction leads to progressive changes, described from the gross appearance, as redness, edema, hyperplasia, fibrosis or hypertrophic changes with keratosis. For simplicity we can diagram this gross pathologic picture and coordinate it with our clinical diagnosis as follows:

GROSS APPEARANCE.		CLINICAL DIAGNOSIS.
Redness ↓ Edema	Reversible	Laryngitis ↓ Contact Ulcer Hyperplasia, Polyp ↓ Vocal Nodule Papilloma
Fibrosis ↓ Metaplasia	Irreversible	Pachydermia Hyperkeratosis Leukoplakia Cancer

CLINICAL RESULTS.

Antibiotic-steriod nebulization has been utilized in the clinical management of all of these conditions. The antibiotic to combat the infectious factor in the etiology of many of these conditions, or the secondary infection that may co-exist; the steriods to act as the blocking or anti-inflammatory agent for the stereotyped reaction that occurs in response to non-specific irritation. In its action it also controls fibrosis of healing.

ACUTE LARYNGITIS.

Case 1. Young male employed in recapping automobile tires developed hoarseness when the overhead ventilator broke down, and he was subjected to the inhalation of concentrated chemical irritants; symptoms had persisted for one week. Examination revealed a diffuse redness of the larynx with edema of the mucosa. Nebulization therapy was recommended. Report of the referring laryngologist 48 hours after therapy was, "improved voice with marked clearing of the local reaction."

ACUTE LARYNGITIS.

Case 2. Young male railroader was seen in consultation because of hoarseness, and painful difficult swallowing; four weeks prior he had been injured and exposed to chemical fumes when a Diesel engine exploded. Treatment during his two weeks of hospitalization consisted of penicillin and confinement in an oxygen tent; at this time the mucosa of the hypopharynx and larynx was red and edematous; there were residual areas of ecchymosis and ulceration in the nasopharynx. One week after nebulization therapy was started the voice had improved, and pain and difficulty in swallowing had subsided. In another week all symptoms had subsided, and the physical findings had returned to normal.

Both cases represent specific therapy for a reversible process.

The contact ulcer has usually been a chronic difficult problem to treat.

Case 1. Music teacher with hoarseness for two weeks, failed to show improvement under a regime of therapy prescribed by her home town laryngologist. Examination of the larynx showed a diffuse edematous ulceration with exudate on the right vocal cord and a concave ulceration of the right arytenoid. One week after nebulization there was 75 per cent improvement; the edema and exudate had subsided, leaving only the concave ulceration of the vocal process; this was larger than on the original examination. Two weeks later healing was complete; voice rest had not been insisted upon.

Case 2. A similar result was obtained in a female patient with hoarseness for two months; excessive talking and smoking had been part of an unusually active social program; there was a large ulceration of the right vocal process. Two weeks after therapy the process was 50 per

cent improved in spite of continuation of her social activity. Because of her apprehension, therapy was continued along with limited voice use. One week later the voice and local findings were normal, and there has been no recurrence to date.

Case 3. S. K., age 42, was a salesman with hoarseness for two years; a laryngoscopy had been performed twice previously for removal of a tumor. Our examination revealed the presence of a large pea-sized granuloma of the right vocal process. A recurrence developed after laryngoscopic removal. Nebulization therapy was prescribed. The patient was not seen again for three months. At this time the voice was good and laryngeal examination revealed complete healing with only a small epithelized nodule at the site of the previous granuloma.

LARYNGEAL POLYP.

The laryngeal polyp is basically a localized edematous reaction. It is referred to by clinicians and pathologists in varying descriptive terminology. Nebulization has been used as the primary agent in many cases, with a satisfactory reversal of the process and improvement in the voice. In other cases treatment has been used as a postoperative adjunct to prevent irreversible granulation. We feel that there have been benefits in both situations.

PAPILLOMA.

Papilloma has been a problem, especially in children, because of its tendency to recur. Improvement with antibiotic therapy has been controversial. We feel that the antibiotic steriod combination has served to abort the process when used as a postoperative adjunct.

Case 1. R. M., age five years, was first seen in 1954. A tracheotomy had been performed at the age of two years, and endoscopic removal of papilloma performed repeatedly. Laryngeal findings at this time were described as a diffuse growth of papillomas in the larynx and extending into the upper segment of the trachea. During 1954 and 1955 we continued to remove papillomas every three months. Nebulization therapy was added to his regime in April, 1955. There was complete arrest of the recurrence, so that in October, 1955, the tracheostomy was closed. There was no recurrence at the last examination in September, 1957. Age at this time was seven years.

Case 2. A. B., age three years, was first examined in 1955 with hoarseness due to a diffuse growth of laryngeal papilloma over both cords. Endoscopic removal was performed at three-month intervals, until February, 1957, when nebulization therapy was started. Papillomas were removed in July, 1957, and there has been no recurrence to date. Patient is now five years old.

Case 3. J. H., age three years, had papillomas removed on three occasions prior to our examination in February, 1957. Nebulization therapy

was started and endoscopic removal performed in February and April. There was no recurrences at the last examination in November.

HYPERKERATOSIS AND LEUKOPLAKIA.

Nebulization has been used both as a primary therapeutic agent and also as a postoperative adjunct in these cases. The progress of therapy can be observed in the routine examination of the patient. We feel that we have been able to reverse the process in cases with early surface changes, and prevent recurrence in those cases requiring endoscopic removal.

TUMORS.

Nebulization has been utilized in the postoperative care of patients with carcinoma *in situ* removed under direct laryngoscopy.

METHOD OF THERAPY.

A No. 40 DeVilbiss nebulizer is prescribed for home use by the patient. The preparation of choice has been cortisporin. This is a suspension of aerosporin, neomysin and hydrocortisone in an aqueous vehicle. Therapeutically this formula combats infection due to susceptible organisms, and relieves symptoms caused by an inflammatory process. The incidence of tissue sensitivity to the components is low. While other preparations may be equally effective, we believe that a suspension offers the advantage of a greater concentration and more prolonged action.

The patient is instructed in the method of nebulization and treats herself four times daily with 6 to 8 inhalations at each procedure. Therapy is decreased to an "as needed regime," with improvement in symptoms and local findings.

SUMMARY.

An analysis and rational for the use of cortico-steriod-antibiotics combination in various disease entities of the larynx has been presented. The clinical results observed in the treatment of various laryngeal diseases with cortisporin

nebulization as a primary agent and as an adjunct to surgery have been reported.

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ANNUAL OTOLARYNGOLOGIC ASSEMBLY UNIVERSITY OF ILLINOIS.

The University of Illinois College of Medicine Department of Otolaryngology announces its Annual Otolaryngologic Assembly from September 29 through October 5, 1958. The Assembly will consist of an intensive series of lectures and panels concerning advancements in otolaryngology, and evening sessions devoted to surgical anatomy of the head and neck and histopathology of the ear, nose and throat. Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Ill

THE ROUTINE BLEEDING AND CLOTTING TIME TESTS: THEIR MEDICOLEGAL STATUS.*†

RUSSELL FLETCHER, M.D.,

San Rafael, Calif.

The reason for presenting a paper on this controversial subject is to review briefly the history of the tests and the reasons why these obsolete "screen" tests are so extensively employed. The modern diagnosis of hemorrhagic disease leaves no doubt that these tests are not accurate; they are not reliable, and actually they may constitute a danger or a menace to the patient and to the physician by inducing false security if he relies upon these tests alone to determine preoperatively whether or not a patient will bleed.

THE HISTORY OF THE BLEEDING AND CLOTTING TIME TESTS.

These tests have been done for 40 years. Bailey[1] in 1922, and Hunt[2] in 1927, each made surveys of several hundred hospitals in the United States in which a large percentage used the tests routinely. They found that most of the hospitals and physicians who had stopped using the tests did so because they were not reliable. Hunt stated that it was not necessary to have tests done if a proper history and physical examination had been done. Shepeard[3] in 1948, stated that the tests were "a vestigial remnant of yesteryear," and the *Journal of the American Medical Association*,[4] in 1955, commented on his article as follows: "There have been numerous reports relating experience from Nose and Throat Departments that show bleeding and clotting time estimates are not practical as tests predicting hemorrhages during and after a tonsillectomy. Normal results are not a guarantee of freedom from hemorrhage, and abnormal results have been ob-

*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Inc., Beverly Hills, Calif., Jan. 18, 1958.

†The blood taken for these tests is taken from a puncture wound of the finger or ear, and is not done routinely by venepuncture.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 4, 1958.

served without subsequent bleeding. Dependence upon bleeding and clotting time tends to lead to a false sense of security. The history and physical examination are much more important, and if such an examination suggests a bleeding tendency, then a thorough study is indicated."

There are some statements in the literature, and in text books, which claim in the same sentence that "although the tests are not reliable, their omission "might have disturbing medicolegal consequences." Some text books have stated that the tests were of value in anticipating hemorrhage pre-operatively. (These indefinite statements and inferences in the literature, are the points that a lawyer grasps in order to convince a jury that one was negligent if the tests were not done). This is illustrated by our experience in Marin County this past year. The surgeons voted unanimously to eliminate the tests pre-operatively; however, the pathologists, who direct all the hospital laboratories, strongly argued that, although they "conceded that the tests are of somewhat limited value," they felt that such statements and inferences as noted above in the literature were of sufficient value for the lawyer to accuse and perhaps convince a jury that there was negligence if the tests had not been done on a patient that subsequently hemorrhaged. The result was that the hospital made it obligatory to have the tests done pre-operatively on every patient.

The following are a few of the outstanding hospitals and clinics that do not have tests done pre-operatively: the Massachusetts Eye and Ear Infirmary, the Children's Hospital in Boston, the hospitals in Baltimore, the University of Michigan Hospital, the Mayo Clinic, the University Hospital in Iowa City, and Stanford Hospital in San Francisco. All of these hospitals had routinely used the tests for years, but abandoned them because they were not reliable.

In a letter from the legal department of the American Medical Association,[5] dated Dec. 13, 1957, it was stated that they knew of no court decision based upon the use or non-use of these tests.

Why are These Tests Still Continued? In spite of the above

information, as well as the fact that practically all physicians concede that the tests are of very little value, why are these tests done in so many hospitals, either optionally on the part of the physicians, or are required by hospital regulations? In a rough survey of the hospitals of the San Francisco Bay area, over 50 per cent of them require these tests preoperatively. In Portland, Ore., seven of the eight hospitals have the tests done.

REASONS GIVEN FOR CONTINUING THE TESTS.

1. An occasional physician "thinks that the tests might be helpful sometime" (rarely is there a physician found who is convinced of this fact). *2.* Some physicians believe that having the tests done reassures the patient. *3.* Many physicians, when asked why they continue the test, answer: "why worry or fuss about eliminating the tests which cost the patient such a trivial or paltry sum" ($2.00 in the San Francisco Bay area); and they add, "anyhow, most patients have insurance." Although economics should never be a consideration in this problem, it is one of the chief arguments of the proponents for continuing the tests. A few facts might be brought out regarding this. If it is not reliable and not necessary, it is wasteful, no matter how cheap it is or who pays for it.

In Marin County, with a population of 30,000, the three hospitals in this county did 5,446 operations in 1957. At $2.00 per test, this amounts to $10,892. As Marin County is approximately 1 per cent of the State of California, this total would be $1,089,200 for the State. If this amount is wasted by requiring an unnecessary test, it is not trivial or paltry. One of the health insurance companies in California that does 10 per cent of the health insurance in this State, handles about 90,000 operations per year; therefore, it is self-evident that elimination of such unnecessary tests would be a material saving to all insurance companies. *4.* The tests have been done for so many years that physicians are hesitant to discard "such time-honored tests," which is chiefly due to inertia. The fact that any test has been done routinely by a large number of physicians in any community was a strong medico-

legal argument for continuing the test. This point is very well covered in the next paragraph, under *"Medicolegal Status."*

5. The Mediolegal Status: The chief reason why physicians and hospitals have continued this routine pre-operative test is the medicolegal threat or intimidation that failure to have had the tests done might be grounds for negligence if a lawsuit were instigated because a patient hemorrhaged. On the Pacific Coast we are particularly aware of malpractice suits. This is reflected in the high rates for malpractice insurance. The rates given by the American Academy of Ophthalmology and Otolaryngology shows that Oregon has the highest rate in the nation, which is about twice as high as California, and Washington which, in turn, are three times as high as the rates in Texas, Pennsylvania or New Hampshire, and twice as high as New York.

What is the accepted or customary practice in the community in which you practice? A letter from the legal department of the American Medical Association to me,[5] dated Dec. 13, 1957, outlines the requirements very well and states in part, as follows: "To comply with legal requirements, a physician must meet the standards of good medical practice in any service he furnishes a patient. These standards are based on what other reputable physicians in the community, or in similar communities, would or would not do in the care of similar cases. One of the tests applied, so far as the use of a particular medical or surgical technique is concerned, is the *general reliability and acceptance of such technique.* If the value of a particular technique is of such a degree that a substantial number of reputable physicians in the community would utilize it in a given situation, then the failure of a physician to utilize it in the same or similar situation would be a difficult factor to explain in a subsequent malpractice suit. It may not constitute negligence *per se,* but the jury would certainly regard such factor with respect. Whether or not bleeding and clotting time tests enjoy the degree of acceptability described above, I cannot say. *That must be decided by the medical profession* . . . I believe a hospital regulation urging routine bleeding and clotting time tests on

all in-patients about to undergo surgery is a sensible one, and that it should not be changed *unless substantial medical evidence is available* as to the *unreliability* and *futility* of *the tests"*; therefore, it is up to the medical profession to show "substantial medical evidence as to the unreliability and futility of the tests."

This evidence is available from three national authorities on hemorrhagic diseases: *1.* The most concise and graphic presentation of this evidence is given in a letter from Louis K. Diamond, M.D.,[6] who is Associate Professor of Pediatrics at Harvard Medical School, and who is in charge of the Blood Clotting Laboratory at Children's Hospital in Boston. He made the following brief survey: *Survey of children referred to the Blood Clotting Laboratory of Children's Hospital of Boston in the past four years:*

Group I. Patients who had had a routine pre-T and A bleeding and clotting time determination in a hospital laboratory where one or both tests were found to be prolonged beyond normal limits and, therefore, were referred to us for more elaborate tests which we found to be entirely normal; nine patients, most of whom later had T and A without any bleeding disturbance.

Group II. Patients who had a definite history of bruising in the past and when explored, frequently a history of bleeding in relatives, but normal bleeding and clotting times had been determined by routine methods; 14 patients, nine of whom had hemophilia of varying degrees and five Christmas disease.

Group III. Patients who bled following surgery, in whom a routine bleeding and clotting time was said to be normal; five patients, of whom four had hemophilia and one Christmas disease.

Group IV. Patients who bled following surgery in whom a good bleeding and clotting time test, even using venous blood, and supervised by our own laboratory, showed normal values, and yet following T and A in seven cases and dental extraction in one case, there was moderate to severe hemorrhage. In two cases blood loss was sufficient to require transfusion.

2. The second authoritative evidence is given by Paul Aggeler, M.D.,[7] Associate Clinical Professor of Medicine at University of California Medical School, and Head of the Blood Clotting Research Laboratory at Children's Hospital in San Francisco. He states that "numerous cases falling into all four categories listed by Dr. Diamond have been extensively investigated in his laboratory. During the past three years alone, 28 patients whose bleeding and venous blood clotting time were normal, were nevertheless proven by more detailed methods to suffer from well defined clotting disorders (hemophilia, P. T. C. deficiency, P. T. A. deficiency, hypoproconvertinemia and fibrinogenopenia). In about one-third of these patients tooth extraction, tonsillectomy, and even major surgery had been performed, with hemorrhagic complications in every instance. In several cases, because of the false assurance afforded by normal bleeding and clotting times, the operation had been done without further investigation, despite the surgeon's complete awareness of the bleeding history. In 20 years' experience in this field Dr. Aggeler can recall not a single instance in which a bona fide hemorrhagic disorder was discovered solely by the use of routine pre-operative bleeding and clotting times. He has seen many cases, however, where false positive results have lead to still further useless and expensive investigations."

3. The third source of evidence that these tests are not reliable, is the article "Diagnosis of Hemorrhagic Diseases—Evaluation of Procedures." Part I appears in the December, 1957, *California Medicine,* and Part II in the January, 1958, issue by L. W. Diggs, M.D., who is Professor of Medicine at the University of Tennessee College of Medicine, and Director of Department of Medical Laboratories, University of Tennessee, City of Memphis Hospitals, who was the guest speaker at the California Medical Association meeting in May, 1957, in Los Angeles. This paper discusses in detail the importance of a careful history and physical examination, and states that "they are more reliable than laboratory tests for the prediction of the tendency to bleed at the time of surgical operation, and failure to obtain a history of bleeding episodes may be catastrophic." He also states that "Routine pre-operative

hemorrhagic studies are not indicated in the case of patients who are not bleeding at the time, or who have a personal and family history negative for abnormal bleeding, and no signs of abnormality at the time of physical examination."

He further states: "On patients whose history or physical examination indicates a bleeding tendency, then special hemorrhagic tests *by experts* are indicated." He stated that "a surgeon would not be considered negligent if he did not perform routine pre-operative tests for bleeding and clotting time." He also states "in the past, the finger tip method was an accepted and standard practice and afforded the surgeon, the laboratory director, and the hospital administration a degree of medicolegal protection which comes from the performance of the usual and customary procedures. Such procedures will not, in the light of present knowledge, continue to be accepted." There are so many interesting and pertinent facts in this article by Dr. Diggs, that it would take too long to review them in this paper. It is, therefore, strongly recommended that everyone read this excellent article by Dr. Diggs.

CONCLUSIONS.

1. The routine bleeding and clotting time tests are not reliable; authorative medical evidence is herewith presented. (It has also been the experience of many clinicians).

2. The tests are dangerous and a menace to the patient and to the physician by inducing a false sense of security.

3. The safest practice for the patient, as well as the physician, is to take a careful history of bleeding in the patient and the family, and to make a good physical examination. If these show any bleeding tendency, then special hemorrhagic studies *by an expert* are indicated.

a. A physician with such a patient will not be guilty of negligence if the routine tests were not done pre-operatively and the patient subsequently bled.

b. A physician with such a patient who relied on the routine bleeding and clotting tests *alone* and did not have special

hemorrhagic tests by an expert, might be accused of negligence.

4. Since the tests have been proven to be so unreliable, they should not be required pre-operatively on surgical patients.

5. "If it is medically sound, it will stand up legally!"[9]

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POST GRADUATE COURSE UNIVERSITY OF ILLINOIS.

The next postgraduate course in Laryngology and Bronchoesophagology to be given by the University of Illinois College of Medicine is scheduled for the period October 27 through November 8, 1958, under the direction of Dr. Paul H. Holinger.

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THE TREATMENT OF POLYPOID LARYNGITIS.*†

GERSON LOWENTHAL, M.D.,

Cincinnati, Ohio.

The term polypoid laryngitis designates a localized or diffuse non-specific inflammatory tumor of one or both vocal cords, involving the epithelium and the subepithelial space of Reinke. This space is a very thin layer of loose connective tissue between the epithelium and the elastic and muscular tissue of the vocal cords.[1] It extends from near the anterior commissure to the vocal process of the arytenoid and to the stria arcuata, superior and inferior,[2] which is the transition point between the squamous and the columnar epithelium above and below. It has a scarce arterial supply but a rich venous plexus. The epithelium is much thinner, only two or three layers, at the free edge and upper surface of the cord, while there are 10 to 20 layers of stratified squamous epithelium over the remainder of the cord.[3] This explains why the inflammatory lesions may be restricted to this space, and why they occur most commonly along the phonating edge and upper surface of the cord.

The most common inciting factors for polypoid laryngitis are considered to be the trauma of vocal abuse[4] or the irritation produced by smoking.[5,6] These lesions nearly always produce hoarseness, tiring of the voice, occasionally pain or discomfort in the throat, and rarely, respiratory embarrassment. Psychologically there is usually apprehension on the part of the patient over the implications of the symptoms.

Clinically, the lesions may be placed in two groups: localized and diffuse. Either of these may be acute, due to sudden vocal strain, or chronic, due to repeated trauma to an unresolved process. In the localized, the tumor may be a pedun-

*Read at the meeting of the Middle Section, American Laryngological, Rhinological and Otological Society, Inc., Chicago, Ill., January 13-14, 1958.

†From the Departments of Otolaryngology, The Jewish Hospital and The College of Medicine, University of Cincinnati, Cincinnati, Ohio.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication March 10, 1958.

culated or sessile mass arising from the free edge or upper surface of the cord, near the junction of the anterior and middle thirds; rarely in the posterior third. Occasionally the lesion may be found in the anterior commissure, and infrequently in the posterior commissure. It may vary in color from a pale glistening gray to a pink, yellow or purple. In consistency it may vary from soft, almost gelatinous, to rather firm and nodular. In the diffuse, the swelling involves the

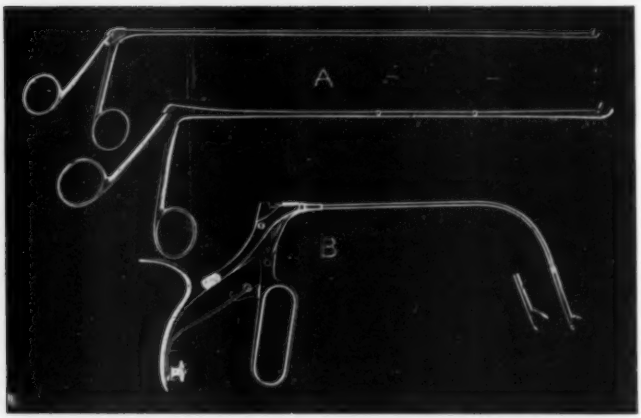

Fig. 1. Instruments used in surgical treatment of polypoid laryngitis. A. Jackson Laryngeal Cup Bite Forceps, Angular Jaw, 2-4 mm. (Direct Laryngoscopy). B. Curved Laryngeal Forceps (Universal Handle) 3-4 mm. (Indirect Laryngoscopy).

anterior half or two-thirds or more of one or both cords. The color may vary from a pearly gray to a dusky pink. This type may also be flabby or somewhat firm.

Histologically, the localized and the diffuse lesions frequently cannot be differentiated and show varying degrees of congestion, edema, thrombosis and hyalinization, described by Friedberg and Segall[3] (see Fig. 4).

New and Erich,[7] in their monumental paper on benign tumors of the larynx, state that "Inflammatory tumors may bear an exact similarity to almost any type of laryngeal

neoplasm, benign or malignant, not only in gross appearance but also in mode of onset and in production of symptoms." They feel that with few exceptions all benign tumors should be removed.

Although the definition, clinical appearance, etiology, histopathology and location may be variable or controversial,

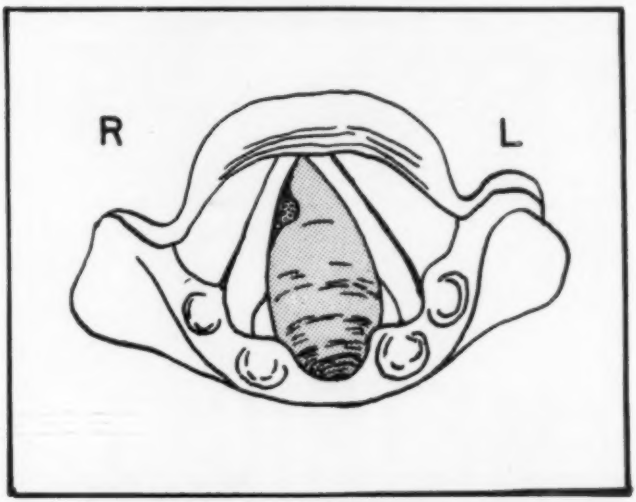

Fig. 2. Most common location on cord—the anterior half (View by indirect laryngoscopy).

there is relative unanimity with regard to treatment. This paper will concern itself primarily with the surgical treatment.

Treatment is divided into three phases: first, the medical or "expectant" phase. A careful survey of the mouth, ears, nose and throat, and a thorough medical examination to rule

Fig. 3. Localized Polypoid Laryngitis. Photograph of a lesion removed from position shown in Fig. 2.

out local or pulmonary, or other systemic disease, is important.[8] Elimination of the inciting causes such as vocal abuse, smoking, and alcohol ingestion, are mandatory. Voice re-education is required in many cases. Specific medical therapy has not been available, although recently steroids have been used by Brodnitz.[9] Some symptomatic relief may be obtained from a bland intratracheal instillation such as Mono-P-

Chlorophenol. If the lesion is reversible within a few weeks, no further treatment may be necessary.

The second phase is surgical treatment, which is essentially an excisional biopsy (see Fig. 3), with gentle, precise, superficial removal of the lesion. It is well recognized that following this principle without damage to the underlying elastic membrane, there appears to be complete regeneration of the cord with normal appearance and function. This has been

Fig. 4. Localized Polypoid Laryngitis. Photomicrograph of specimen shown in Fig. 3, demonstrating congestion, edema, thrombosis and hyalinization of the loose subepithelial connective tissue.

demonstrated by Jackson, Tucker,[11] Lore,[10] Holinger[12] and others.[13] Jackson[15] states that it is better to leave a convex edge of the lesion on the cord, even if this should allow a little of the basal tissue to remain, rather than to remove too much tissue, leaving a concavity. If necessary, a secondary procedure can be done. In polypoid lesions with a suggestion of hyperkeratosis or leukoplakia, biopsies must be taken from various areas in the larynx and carefully labeled as to the site of origin of each specimen. Thus, early or *in situ* carcinoma can be localized.[14]

The recognized methods of surgical removal are by indirect[5] or direct[12] laryngoscopy under local or general anes-

thesia, or by suspension[7,19] laryngoscopy under general anesthesia. Rarely, an external approach with tracheotomy may be necessary for removal of a large growth[16] The surgical treatment in the series of cases analyzed was by indirect laryngoscopy, direct laryngoscopy, and most frequently by a combination of the two, under local (topical) anesthesia. The operation is simple, orderly, and relatively easy on the patient. For a successful procedure it is necessary to secure the cooperation of the patient, and to obtain good anesthesia.

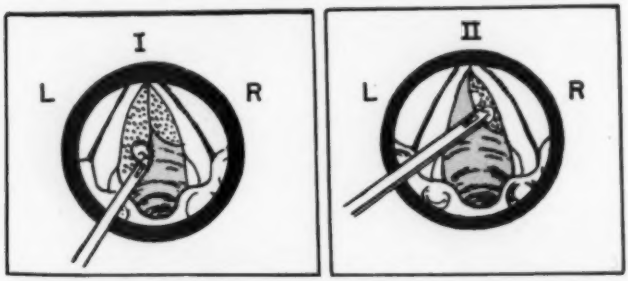

Fig. 5. Diffuse Polypoid Laryngitis, Bilateral. Histologically these lesions are similar to the one shown in Fig. 4. I and II—Steps in the surgical treatment (Direct laryngoscopic views).

Since the lesions under discussion occur most frequently in adults, this can usually be accomplished.

Premedication consists of seconal or nembutal, grains $\frac{3}{4}$ to grains $1\frac{1}{2}$, taken at bedtime the night before surgery and repeated one hour pre-operatively. Morphine sulfate, grains 1/8 to grains 1/6, and scopalomine, grains 1/300 to grains 1/200, are given hyperdermically one-half hour pre-operatively.

The mouth, oropharynx and hypopharynx are sprayed with 2 per cent pontocaine solution two or three times at about

three-minute intervals, and the patient is encouraged to expectorate the excess. Some of the anesthetic is applied to the upper gum to reduce the discomfort of the laryngoscope later.

Cocaine hydrochloride, 5 cc. of 10 per cent solution, is freshly prepared in the operating room from a measured amount of crystals. This is instilled into the laryngopharynx and larynx by the drop method. Using a laryngeal syringe with a curved tip, several drops are placed into the valleculae and into each piriform sinus. The patient is instructed to breathe quietly while the tongue is being held, after which he expectorates the excess and the procedure is repeated. One cc. is then instilled over the cords with the patient saying "A" or "E" for a few moments. This is also repeated in a few minutes. Rarely is more than 3 or 4 cc. of the solution required.

The combined technique is used most often. First, by indirect laryngoscopy the lesion is removed by evulsion with a double cupped biting forceps. The patient is usually unaware that this maneuver is different from the instillation of the anesthetic. Depending upon the size of the lesion, a large or small tip is used. Most, if not all, is removed by this technique.

Secondly, a direct laryngoscopy with a Jackson or Holinger anterior commissure laryngoscope, using the Andrews chest support, or more recently, the Lewy[17] holder, is performed with the patient in the supine position, placing a folded blanket under his shoulders. Direct laryngoscopy allows closer inspection of the lesion, and permits trimming of small, loose fargments of mucosa with a small angulated cupped forceps.

Multiple and diffuse lesions are removed except when the anterior third of both cords are involved; (see Figs. 2, 3, 4); then, the more severely involved side only is operated upon. The polypoid cord on the other side is compressed or pinched with the double cupped forceps in two or three areas to reduce the edema. This may be a form of superficial scarification (see Fig. 5). It was noted that in a number of patients a subsequent procedure on the second cord was obviated by this

technique. *Tschiassny[18] states that scarification is an old treatment and attributes the idea to Hajek. Waldapfel[1] reported the use of "simple, superficial scarification of flabby tissue" in such a case, with complete resolution of the lesion without visible scar formation.

The third phase is post-surgical. This consists of complete vocal rest for a period of 10 to 14 days, followed by a regime of sparing the voice for a period of several weeks and by voice correction therapy, if indicated. This phase also includes the removal of the factors which predisposed to the lesion initially. At the conclusion of the third phase the cords appear to be well healed and the voice returns to normal. Recurrences are uncommon.

The case histories reviewed for this paper are of patients over 20 years of age who failed to respond to the medical or "expectant" treatment described in phase one and, therefore, required surgical intervention. This group consisted of 62 adult private patients operated upon for benign laryngeal tumors during the past ten years. It is interesting to note that even in this small series there was a general distribution of lesions comparable to the vastly larger series reported by New and Erich,[7] and by Holinger.[12] Polypoid tumors predominated in the small group, as they did in the larger series. Thirty-five of the lesions were polypoid tumors, of which 26 were localized polyps and nine showed diffuse polypoid degeneration of both cords. Six were papillary tumors; six were multiple papillomata; there were three angiomas; one hemangioma; two granulomas; three amyloid tumors; one leukoplakia; two cysts; two thrombosed varices and one vocal nodule. In six instances other than the papillomata there were multiple localized lesions, sometimes of different types.

In spite of the variation of diagnoses of these benign tumors and of their neoplastic and non-neoplastic character, the treatment of this entire series was essentially the same. Following surgery the cords healed well and appeared normal, and the voice returned to normal within a period of several weeks. In only two cases was it necessary to go back a second time

*He suggests that diffuse lesions be known by the descriptive term "subepithelial chronic edematous corditis."

to remove further tissue. In one instance there was a recurrence of a lesion on the same cord three years later. The second lesion, however, occurred at the junction of the middle and posterior thirds, whereas the original was at the junction of the anterior and middle thirds. The lesions also varied somewhat, the first being a polyp and the second a cyst. In nine patients with bilateral diffuse polypoid degeneration of both cords, only three required operation on the second side after use of the compression (scarification) technique. In one, with only slight diffuse involvement of the second cord, the compression technique was not necessary, and this patient also made a good recovery.

SUMMARY AND CONCLUSIONS.

1. The particular structure of the subepithelial space of Reinke predisposes to the formation of laryngeal polyps on the vocal cords.

2. In the series presented, as in other series, the most common benign tumors are inflammatory, and of these the localized polyps are the most frequent.

3. Treatment is divided into three phases: the medical or "expectant"; the surgical, wherein under local (topical) anesthesia, combined indirect or direct laryngoscopy is performed in all cases; the post-surgical, with removal of the initial predisposing factors and voice correction therapy if required.

4. In diffuse polypoid involvement of both cords, the larger lesion is removed. The smaller lesion is compressed (superficially scarified) in two or three areas with a double cupped forceps. This frequently obviates the need for surgery on the second cord.

5. In general, the prognosis in these lesions is good. Recurrences are uncommon.

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280 Doctors Building.

SUBCOMMITTEE ON HEARING IN CHILDREN.

The American Academy of Ophthalmology and Otolaryngology, through its Subcommittee on Hearing in Children of the Committee on Conservation of Hearing, has been conducting a long-term nationwide study of problems relating to the conservation of hearing in children. The specific aims are to develop the most efficient case-finding methods and to use these methods in estimating the magnitude of the problem in the country; to study state laws and review current practices and facilities for rehabilitation of hearing impaired children; to help develop methods for medical and surgical rehabilitation standards; and ultimately to use the Subcommittee findings in assisting professional workers to improve and enhance programs in hearing loss.

In the second year of operations, a full-time Executive Director has been engaged, and offices established at the Graduate School of Public Health, University of Pittsburgh. An initial study is being conducted in Pittsburgh to identify early medical signs and symptoms which may indicate danger of hearing impairment, to measure the psychological, social and other effects of such impairment and to develop efficient and economical methods for the testing of hearing in children. The Pittsburgh study is a cooperative effort among the following: The Subcommittee on Hearing in Children, the Graduate School of Public Health and the School of Medicine of the University of Pittsburgh, the Pittsburgh Board of Public Education, and the Allegheny County Department of Health.

The members of the Subcommittee on Hearing in Children are: Dr. John E. Bordley, Baltimore; Dr. Victor Goodhill, Los Angeles; Dr. Hollie E. McHugh, Montreal; Dr. S. Richard Silverman, St. Louis; and Dr. Raymond E. Jordan, (Chairman) Pittsburgh. An advisory committee of consultants from the University of Pittsburgh includes Dr. Samuel M. Wishik, Dr. Leo G. Doerfler, and Dr. Isidore Altman. Grants from the United States Children's Bureau through the Pennsylvania Department of Health and from the National Institutes of Health are providing financial support.

PROGRAM OF THE SEVENTH INTERNATIONAL CONGRESS OF BRONCHOESOPHAGOLOGY.

Meeting Place—Kyoto University, Kyoto, Japan.

Friday, September 12th, 1958

8:00 P.M.-10:00 P.M.—Reception (Party) Shimomura House in Kyoto.

Saturday, September 13th

8:00 A.M.—Inaugural Session (in Kyoto University Hall).

9:00 A.M.-12:00 M.—First Scientific Session.

12:00 M.-2:00 P.M.—Luncheon (Reception. The Place not decided).

2:00 P.M.-6:00 P.M.—Sightseeing in Kyoto.

8:00 P.M.—Banquet, Miyako Hotel.

Sunday, September 14th

8:00 A.M.-11:30 A.M.—Second Scientific Session.

11:30 A.M.-12:00 M.—Closing Ceremony.

12:00 M.—Departure for sightseeing in Nara.

1:00 P.M.—Luncheon, Nara Hotel.

2:00 P.M.-6:00 P.M.—Sightseeing in Nara.

7:00 P.M.—Return to Kyoto.

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(Secretaries of the various societies are requested to keep this information up to date).

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Place: The Homstead, Hot Springs, Va., March, 1959.

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Annual Meeting: October, 1958, Chicago, Ill. (Definite time and place to be announced later).

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Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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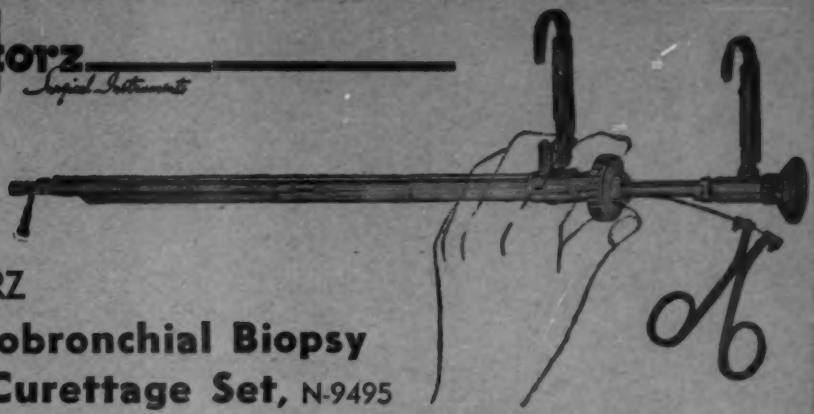

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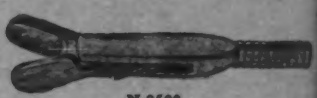

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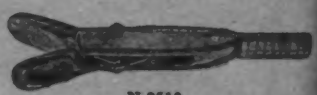

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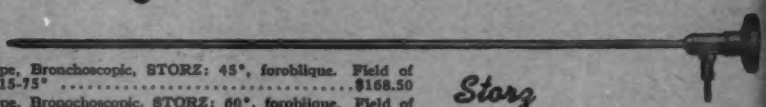

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1. Orzac, E.: Medical Care of the Child Patient Before and After Adenoidectomy and Tonsillectomy, N.Y. State J. Med. 56:886 (Mar., 1956).
2. Ersner, M.S. and Lerner, S.S.: M. Clin. North America 40:1749 (Nov., 1956).
3. Peele, J.C.: Adrenosem in the Control of Hemorrhage from the Nose and Throat, A.M.A. Arch. of Otolaryng. 61:450 (Apr., 1955).
4. Dennehy, P.J.: The Care of the Prostatic Cavity, South African Med. 30:21 (Apr., 1956)
5. Brown, W.S.: The Use of Adrenosem Salicylate to Control Postoperative Bleeding in Plastic Surgery: Dermabrasion, Northwest Med. 57:470 (Apr., 1958)
6. Perkins, R.E.L.: A Clinical Investigation of Adrenochrome Monosemicarbazone Sodium Salicylate, Oral Surg., Oral Med., Oral Path. 10:230 (Mar., 1957).
7. Brode, A.B. and Chianese, T.C.: A Clinical Evaluation of Adrenosem Salicylate. Ann. of Dentistry 15:56 (Sept. 1956).
8. Zubieta C.B. and Escanaverino, R.B.: Adrenosem for the Prevention of Bleeding in Tonsillectomy, Am. Pract. 3:385 (Mar., 1957).
9. Riddle, A.C., Jr.: Adrenosem Salicylate: A Systemic Hemostat, Oral Surg., Oral Med., Oral Path. 8:617 (June, 1955).

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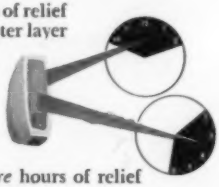

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Notice of each book or monograph, including title, name of author, publisher, pagination, price, etc., will be presented in these notices so that our readers may have all data at their disposal for further inquiry.

ANAESTHESIA FOR NURSES. By Eric Godwin, L.R.P.C., M.R.C.S., F.F.A., R.C.S., Consultant Anaesthetist to the Croydon Group of Hospitals. 98 pages with Index and numerous illustrations. John Wright & Sons Ltd., Bristol, England. 1957. Price $2.50.

CLEFT PALATE AND SPEECH. By Muriel E. Morley, M.Sc., F.C.S.T., Speech Therapist-in-Charge of the Speech Therapy Unit, the United Newcastle upon Tyne Teaching Hospitals and the Newcastle upon Tyne Hospital Management Committee Group. Fourth edition. 271 pages with Index and 86 illustrations. E. & S. Livingstone, Ltd., Edinburgh and London. 1958. Price $6.50.

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*Council on Pharmacy and Chemistry: New and Nonofficial Remedies, Philadelphia, J. B. Lippincott Co., 1956, p. 505.

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